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1959

HAWAII
MEDICAL JOURNAL
AND
INTER-ISLAND NURSES' BULLETIN

**ANNUAL
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**TABLE OF CONTENTS
PAGE 314**

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PREGNANCY SALVAGE WITH NORLUTIN

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Total number of pregnancies	297	45
Number of pregnancies salvaged	46	19
Percentage of pregnancies salvaged	15.5%	42.2%

Adapted from Hodgkinson *et al.**

Indications for NORLUTIN: Conditions involving deficiency of progesterone, such as primary and secondary amenorrhea, menstrual irregularity, functional uterine bleeding, endocrine infertility, habitual abortion, threatened abortion, premenstrual tension, and dysmenorrhea.

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*Hodgkinson, C. P., Igna, E. J., & Bukeavich, A. P.: *Ann. New York Acad. Sc.* 71:753, 1958.

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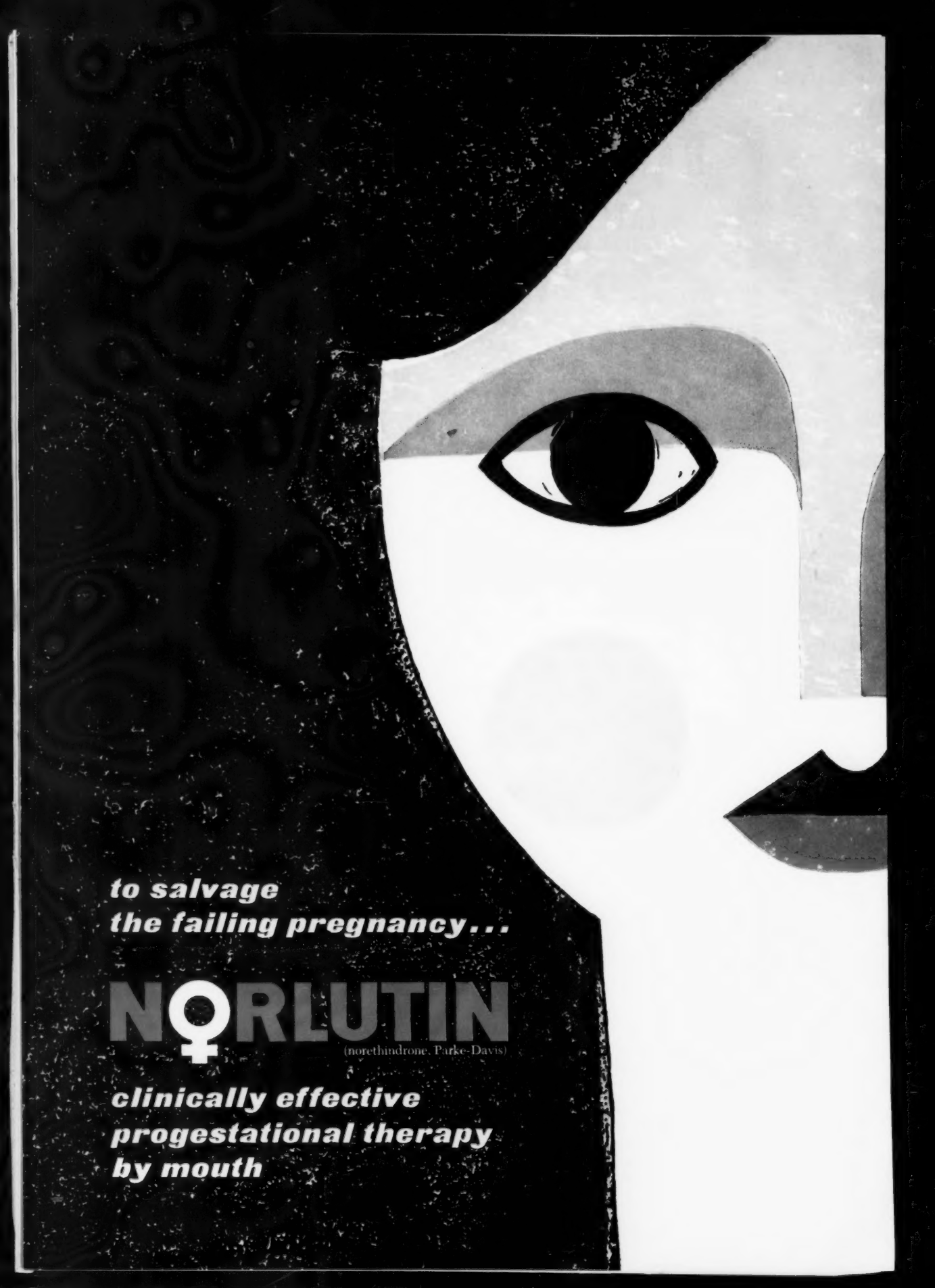
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by mouth

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Correspondence

PAT ON THE BACK

TO THE EDITOR:

Sorry we are so late in sending in our subscription fee. Enclosed please find check for same.

We enjoy your JOURNAL very much, you put out a very good one.

Please mail direct to me.

Sincerely,
H. ELLWOOD HELLING, M.D.
Editor
Jan. 14 Lawrence County Medical Society

"FEE SPLITTING" BY GROUPS

TO THE EDITOR:

I read with interest your editorial on fee splitting. You state that it is "unrealistic" for the American Medical Association to condemn as unethical this practice insofar as groups are concerned because the majority of groups do so split the patients' fee. By the same logic if a majority of the referring and consulting physicians in private practice likewise split the patient's fee, then it would be equally unrealistic for the AMA to condemn this as unethical. The confusion here arises from what I call the principle of aggregates which may be stated so "Any action which is unethical or immoral when committed by one or two people becomes a moral and ethical one when committed by large numbers of people or by one person on a large scale." Examples: If one man kills another, this is murder, and his crime is punished by death. If one man kills 50,000 men by the use of some instrument such as an atomic bomb, his action is a patriotic and noble one and he is to be commended. . . . If one physician splits a fee with another, this is unethical, but if he splits his fee with 20 other physicians then his action becomes an ethical one.

The problem of fee splitting throws many interesting lights on group practice. I have never heard of a group physician referring a patient to a specialist outside of his group when that specialty is represented within his group. When one of my patients needs the services of a consultant, I ask him the following double barreled question. "Do you know of any doctor that you would like to see, or would you prefer for me to recommend one to you?" So here you have an interesting anomaly. The "captive patient" of Grove Farm Co.'s "closed medical panel" has "free choice" of consultants whereas the "free patient" who goes to a group becomes a "captive patient" when he has need of a consultant.

The AMA has calculated that the average general practitioner is able to take care of from 85 to 87% of the patients that he sees. So it can be easily seen that even though every patient who is referred to another physician were a source of income from split fees for the referring physician, the portion of the physician's income derived from split fees would be a minor one in comparison to his total income. This is in contrast to the group physician whose total income is derived from split fees. It is said that a split fee is a bad practice because it encourages the referring physician to send his patient not to the consultant on the basis of his medical ability, but on the basis of the economics involved in the split fee. If this is true for the private physician, how much more true can it be for the group physician, whose total income is derived from split fees?

(Continued on page 422)

D

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Contents

Scientific Articles

Peritonitis in Infants and Children ..... James L. Mertz, M.D. 341
Hemolytic Disease of the Newborn Due to ABO Incompatibility ..... S. H. Waxman, M.D. 344
Some Aspects of Physiology in the Premature Infant ..... Ross Y. Hagino, M.D. 349
Diagnosis and Management of Fibrocystic Disease of the Pancreas ..... Nasser Movassaghi, M.D. 358
Liver Function in Infants and Children ..... Richard K. B. Ho, M.D. 362
Pulmonary Hyaline Membrane Disease in the Newborn ..... John A. Harbinson, M.D., and Kinsuke Nishimura, M.D. 367
Fetomaternal Transfusion Syndrome ..... Donald F. B. Char, M.D., and George M. Ewing, M.D. 372

Editorials

Impressions of a Visiting Professor at the Kauikeolani Children's Hospital in Hawaii 339
Kauikeolani Hospital Jubilee 375
"Operation Hypo" 375
Hawaii Society of Internal Medicine 376

Features

Book Reviews 383
Correspondence 313
County Society Reports 388
Hawaii Medical Association 384
HMSA 380
Honolulu County Medical Library 382
In Memoriam—Doctors of Hawaii—XIX 381
Notes and News 386
President's Page 374
This is What's New! 377

Inter-Island Nurses' Bulletin

Rosie Chang, R.N., M.Litt., *Editor*

President's Message 392
Editorials 393
District and Section News 393
Bylaws 395

Medical Technologists' Bulletin

..... 404

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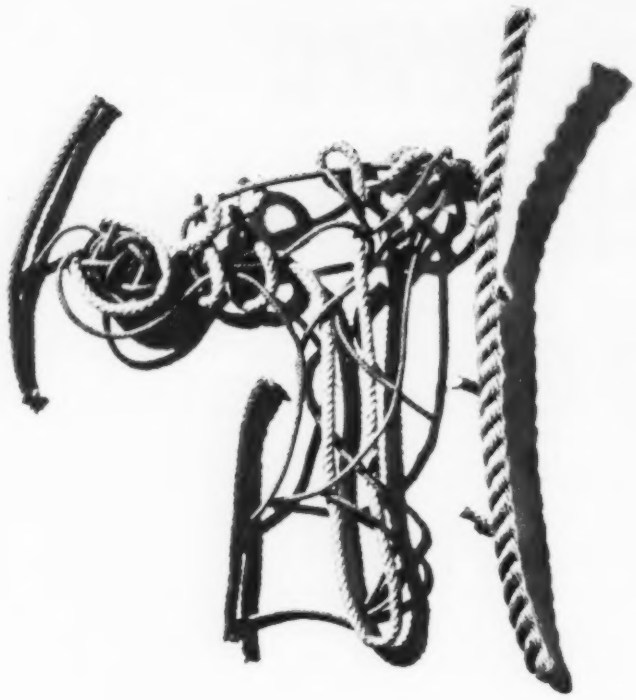

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Investigator or Study	No. of patients	Improvement		
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Bunim, J. J., and others[3]	18	5	7	6
Series A[5]	15	4	9	2
Series B[5]	6	6	—	—
Series C[5]	3	3	—	—

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		Excellent	Fair to good	Poor
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Series F[5]	20	13	4	3

clinically preproved in steroid-responsive diseases

IMPROVEMENT WITH DERONIL IN A WIDE VARIETY OF ALLERGIC AND INFLAMMATORY SKIN DISEASES[5]

Disease	No. of patients	Improved	Same	Worse
Seborrheic psoriasis	1	1		
Neurodermatitis	5	5		
Allergic dermatitis	5	5		
Psoriasis	5	2	1	2
Lupus erythematosus, chronic discoid	1		1	
Atopic dermatitis	3	3		
Acne rosacea	1		1	
Nummular eczema	2	2		
"id" reactions	2	2		
Contact dermatitis	2	2		
Pityriasis rosea, severe	1	1		
Urticaria, chronic	1	1		
Totals	29	24	3	2

THERAPY WITH DERONIL IN A VARIETY OF INFLAMMATORY EYE DISEASES[5]

Patient, age and sex	Diagnosis	Symptoms	Results with DERONIL	Side effects
A.B., 46, M.	Postoperative uveitis		Improved; treatment being continued	None reported*
A.E., 53, M.	Choroiditis	Severe choroidal involvement	Excellent; marked improvement	None*
B.F., 60, F.	Acute choroiditis	Marked visual loss and choroidal effect	Marked improvement; therapy being continued	None*
B.K., 29, F.	Chronic uveitis	Generalized involvement	No change despite dosage increase	None*
H.K., 28, M.	Acute iritis	Blurred vision	Excellent; recovered	None*
A.P., 52, M.	Uveitis and perivasculitis	Vision loss to 20/80	Marked improvement; vision 20/30; treatment continued	None*
A.S., 34, M.	Uveitis		Excellent; patient recovered	None*

*Short-term therapy (AMA 12/30)

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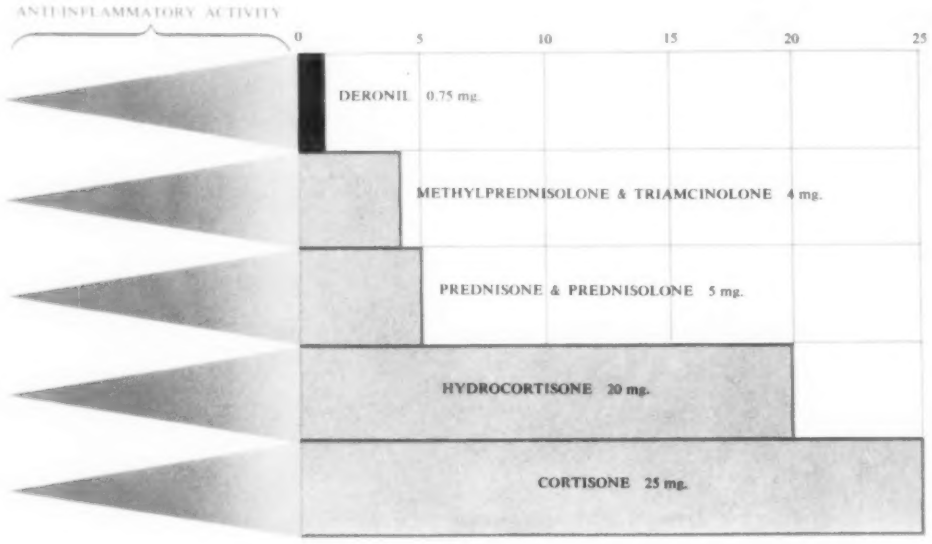

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Bibliography

(1) Boland, E. W., and Headley, N. E.: Preliminary clinical observations with a new series of synthetic corticosteroid compounds in patients with rheumatoid arthritis, Paper presented at Annual Meet., Am. Rheumat. Assn., San Francisco, June 21, 1958. (2) Boland, E. W.: California Med. *88*:417, 1958. (3) Bunim, J. J., and others: Arthritis and Rheumatism *1*:313, 1958. (4) Spies, T. D.; Stone, R. E., and Niedermeier, W.: South. M. J. *51*:1066, 1958. (5) Reports to Clinical Research Division, Schering Corporation.

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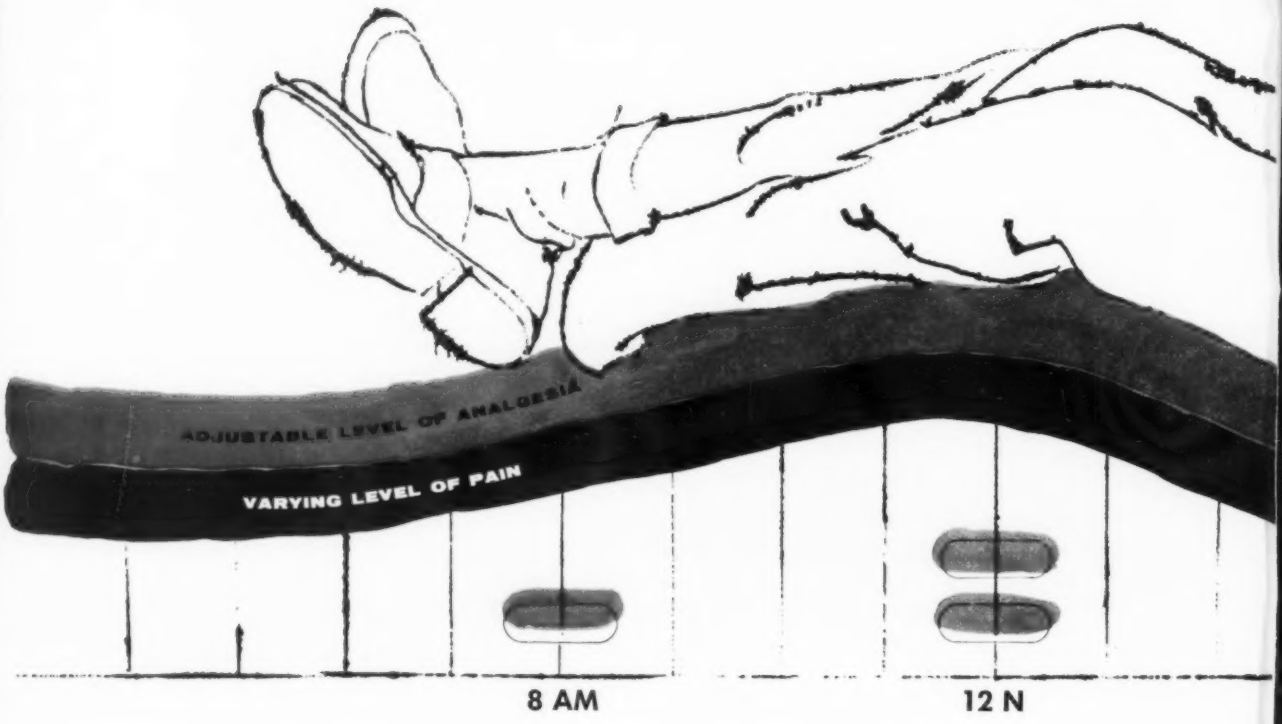

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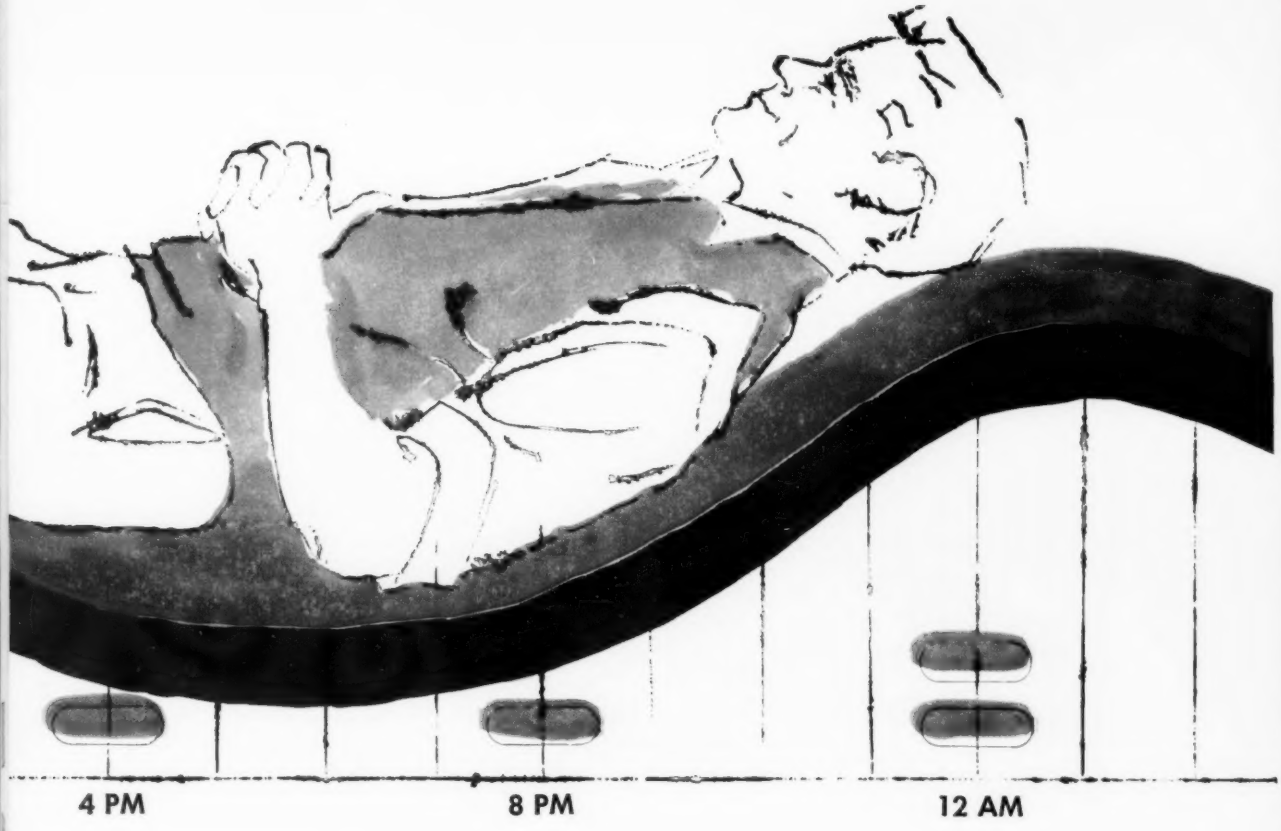

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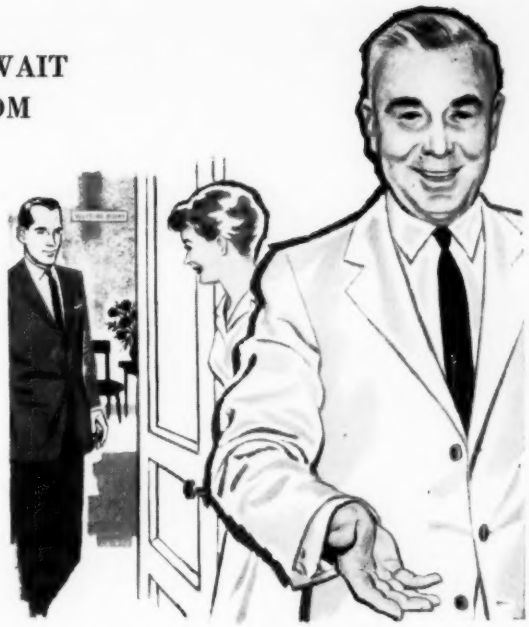

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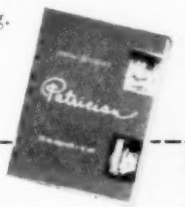

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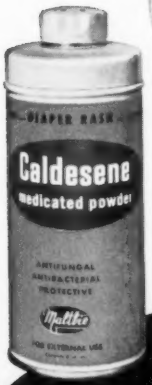

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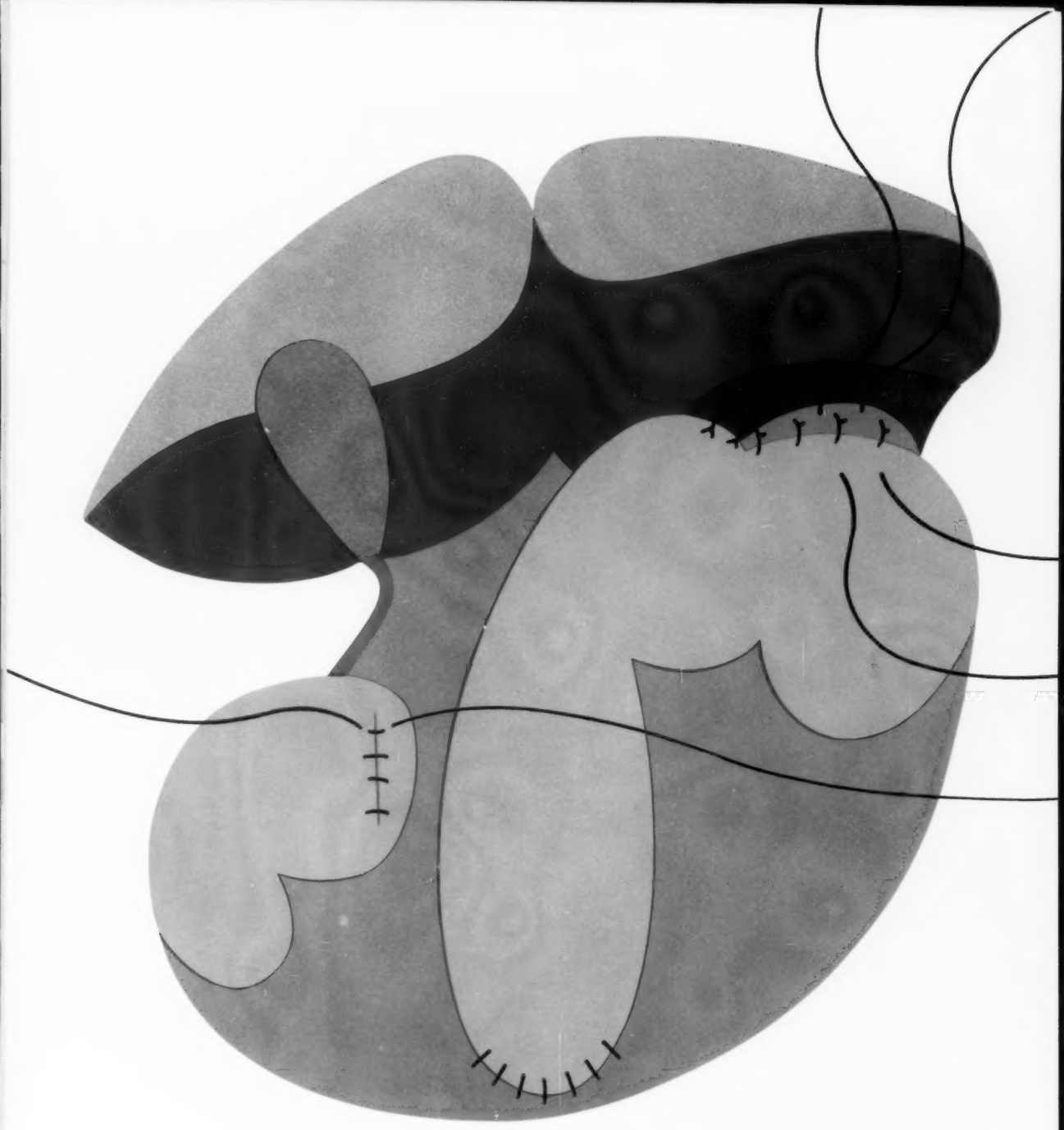

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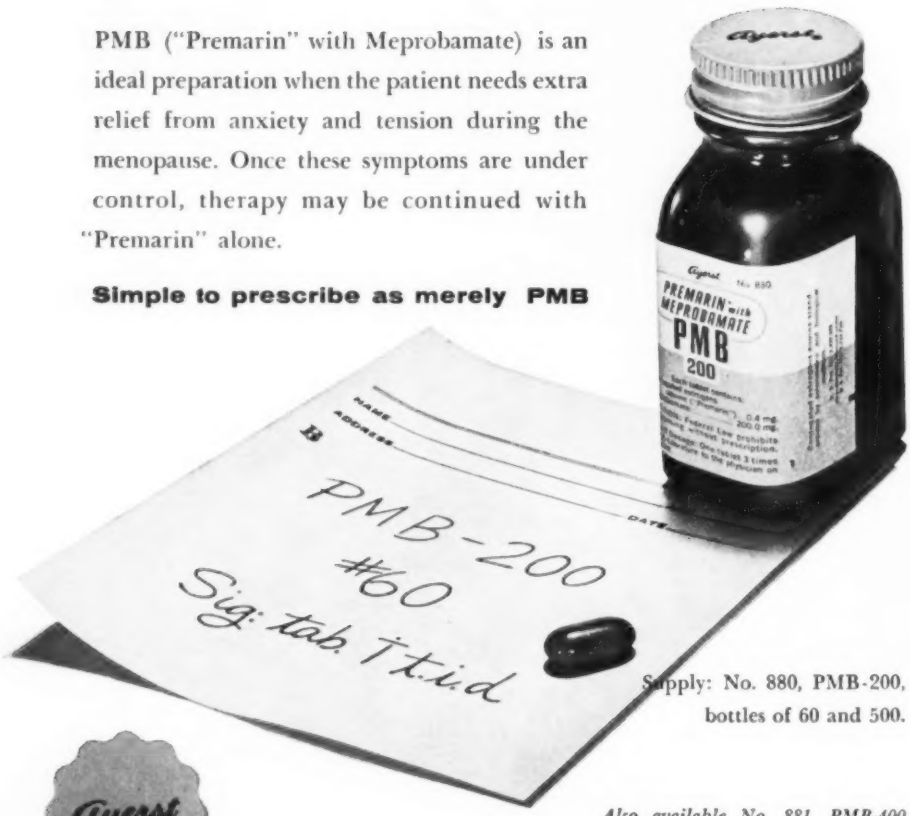

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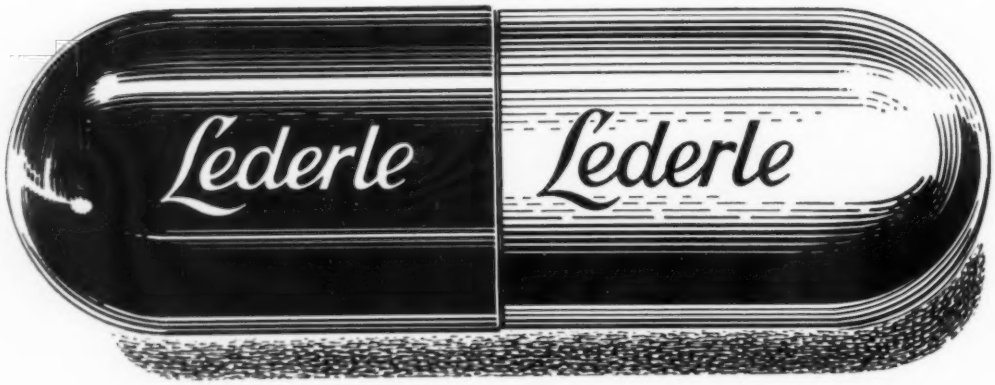

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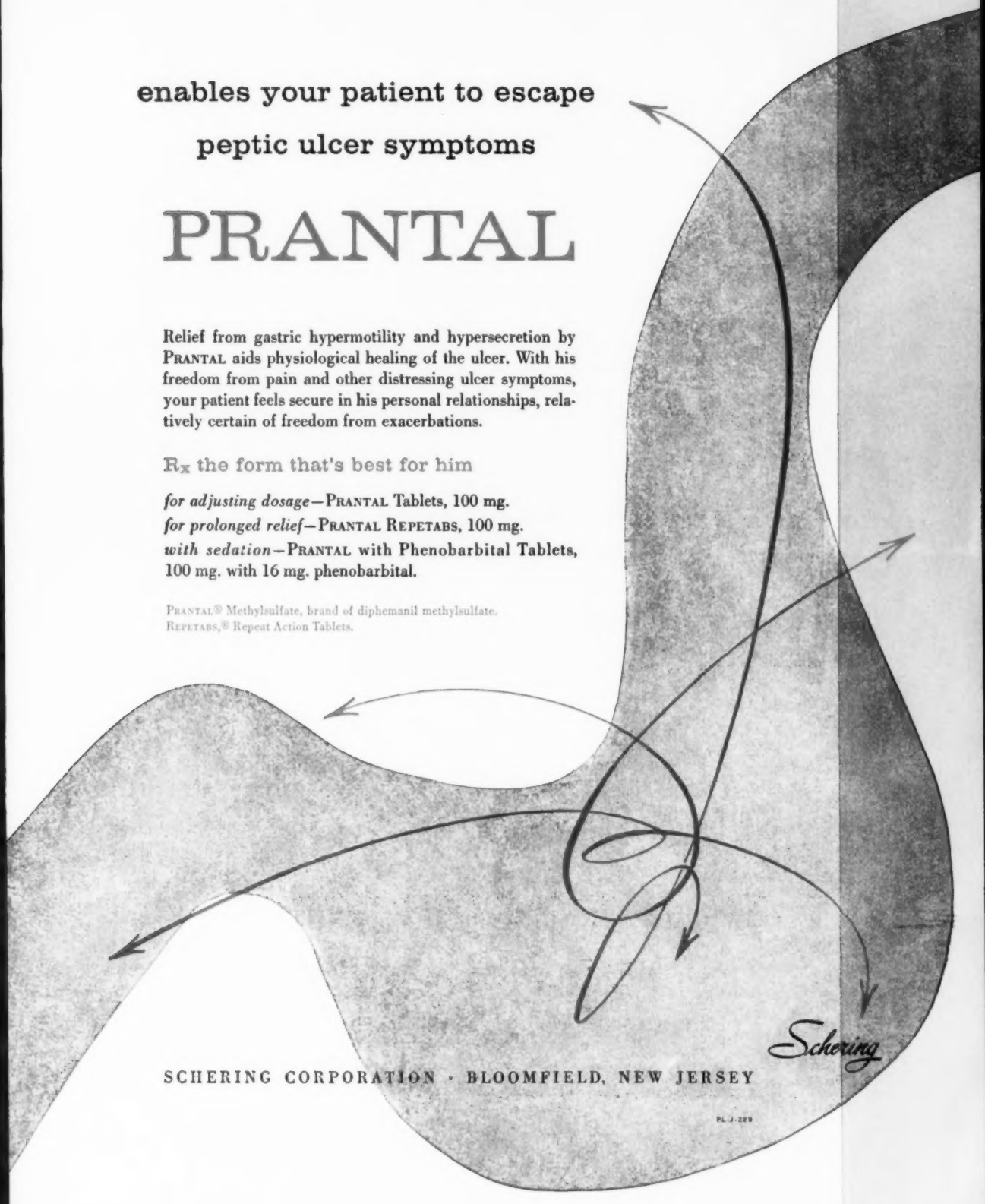

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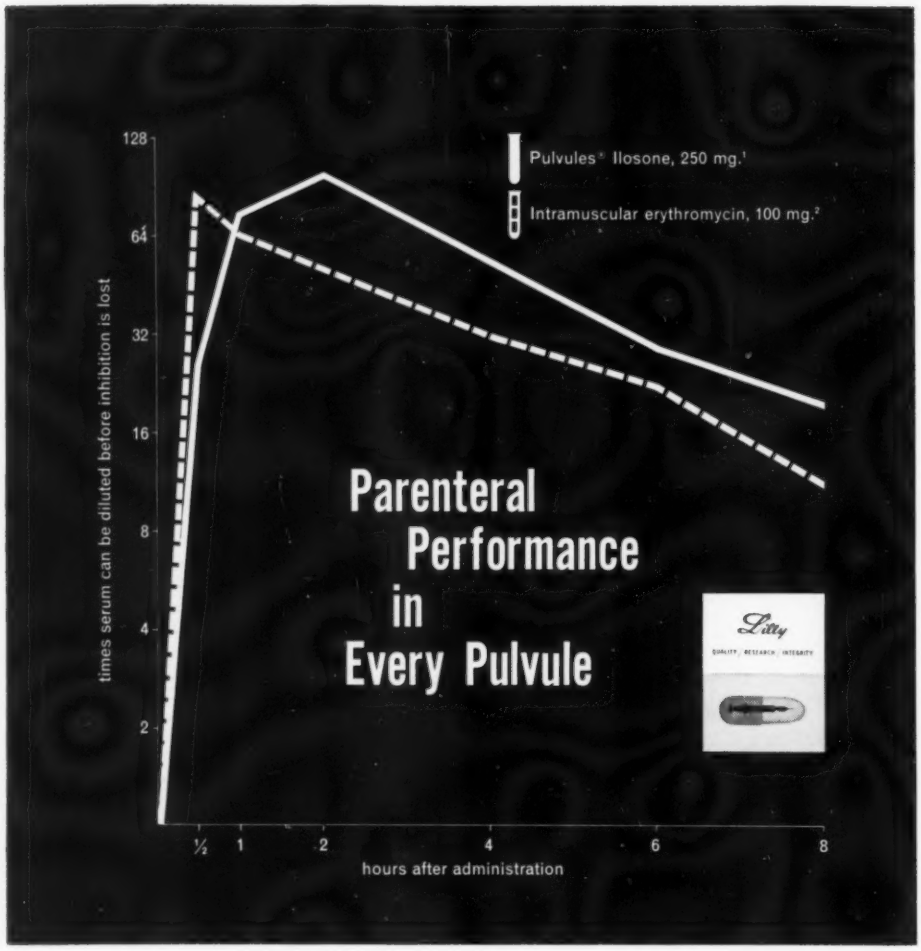

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1. Antibiotic Med. & Clin. Therapy, *5*:609, 1958.
2. Data from Antibiotics Annual, p. 269, 1954-1955.

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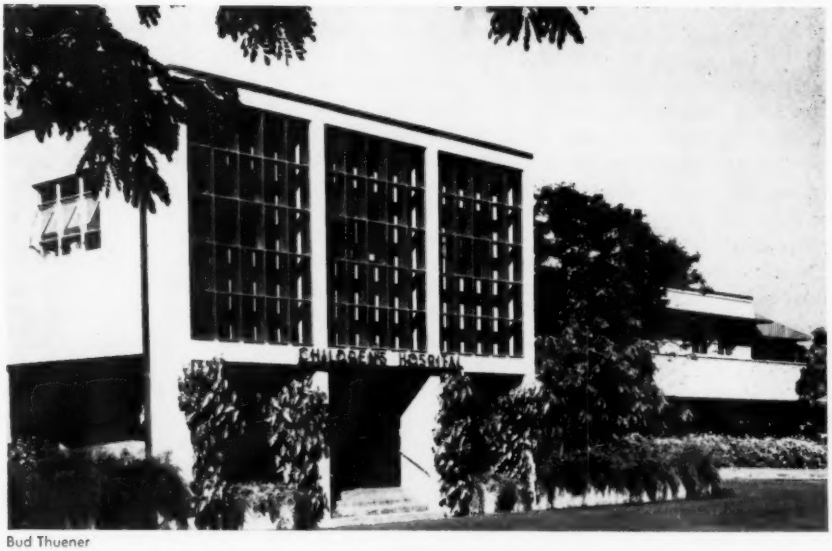
Guest Editorial

Impressions of a Visiting Professor at the Kauikeolani Children's Hospital



Bud Thuener

PAUL GYORGY, M.D., M.D.h.c.,* Philadelphia, Pennsylvania

It should be rightly deemed presumptuous to put in print impressions gained in four weeks of observations and direct personal experience. For any newcomer, it is obvious that early impressions could not be rooted deeply in the soil and may lack the accumulated wisdom which only prolonged, critical, and balanced study may build up. On the other hand, as physicians we are trained in quick assessment of a given medical situation. With accumulated, matured practical acumen pitfalls in diagnostic approach are often recognized and discarded. Thus, we become accustomed to shortcuts, not only in our medical judgment but in many other situations.

This explanation is given as a kind of apology not only for the following subjective views on the state of medical profession and education in Honolulu seen from the angle of a Visiting Professor at the Children's Hospital, but also for the projection in the future of medicine in Hawaii, soon the 50th State of the Union.

It should be considered an axiom, accepted by all progressive clinicians and medical educators, that the best hospitals are and should be supported from three equally important sources; (1) patient care, (2) teaching, and (3) research. Only a successful synthesis and coordination of the above three parts will ensure healthy, enduring growth of a hospital in its entirety.

DR. GYORGY

The foundation for this development, the first in the Territory of Hawaii, was laid by the appointment in 1956 of Professor Irvine McQuarrie as Director of Medical Education on a full-time basis. By the end of 1957, an excellent intramural medical education program was in full swing, very ably and efficiently conducted, after the withdrawal of Dr. McQuarrie, by Dr. Donald F. B. Char. Visiting professors and guest lecturers were and are an integral part of the program as very succinctly sum-

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marized in a previous editorial of this journal by one of the past visiting professors, Dr. Alex J. Steigman.† The present visiting professor fully subscribes to Dr. Steigman's statement that the educational program as carried out at the Kauikeolani Children's Hospital, "could well serve as a pattern for any children's hospital lacking the advantage of medical school association."

But beyond that, the various visiting professors and guest lecturers often differ in their views in many medical programs. This sometimes perhaps slightly confusing fact will at least counteract to a healthy extent the development of dogmatism, to which young residents and sometimes even older colleagues in practice are often only too prone to succumb. This is an advantage of the program, which puts it above that in one given hospital association with a medical school.

The most welcome and unexpected surprise the present visiting professor had was the pleasure to experience the excellent attendance by pediatricians, general practitioners, and even some other specialists at the various rounds and seminars. There must be a real, sincere desire and spiritual need for education and knowledge, much more so than in the large cities of the Mainland. On clinical rounds the excellent, up-to-date training, devoted interest in their patients by the attending physicians became clearly evident.

The question arises: how might this fortunate beginning, still limited to the Children's Hospital and the pediatric specialty, be enlarged and extended?

First, the program at the Children's Hospital should be completed by the addition of an independent, but closely coordinated research foundation under the full-time direction of a highly qualified young pediatrician with academic-clinical and research training. It testifies to the foresight of the Medical Staff of the Children's Hospital and of the Board of Trustees that such a plan is very actively discussed and pursued at the present time.

The director of the research foundation should receive an academic appointment at the University of Hawaii, perhaps as the first member of a Postgraduate Medical Faculty. Enlargement of the faculty could then easily branch out into other hospitals and other specialties on an over-all coordinated basis.

In postgraduate medical education, under the leadership of the American Medical Association, the Association of Medical Colleges and other leading medical societies, more and more emphasis is put on teaching in basic science. This not only for residents, but also for physicians in practice. Hawaii lacks at present a full complement of teachers in basic science, at least in its relation to medicine. Professors of Anthropology, Genetics, Chemistry (Biological Chemistry), Physics, Microbiology, and other related ancillary specialties may be available among members of the University of Hawaii. Anatomy, Physiology, Pathology, Pharmacology, and others are, however, probably not or not fully adequately represented. On the Mainland there is an increasing tendency to create new two-year medical schools in connection with colleges or universities with good laboratory facilities and at least related staff in basic science. Most of the four-year medical schools in large cities could easily assimilate students for the clinical years but are unable to enlarge their "basic science" facilities in the first two years of the medical curriculum. A two-year medical school in Hawaii would fulfill this need, not only for Hawaiians, but even more for students from the Mainland. It would serve also as a link to the Orient and would at the same time provide a complete faculty in basic medical science for postgraduate medical education. The plan seems perhaps too ambitious but it is sound, and with good will and forceful initiative, it should be realized.

† Steigman, A. J., Hawaii Med. J. 17:333 (Mar.-Apr.) 1958.

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Scientific Articles

Surgery is generally indicated for peritonitis in children. Rarely, it may even save a secondary nonappendiceal case.

Peritonitis in Infants and Children

JAMES L. MERTZ, M.D.,* Honolulu

DURING THE YEARS 1943 to 1958 at Kauikeolani Children's Hospital there have been recorded 61 cases of peritonitis. Of these, 14 were of the "primary" or "idiopathic" type in which no focus of infection could be demonstrated within the peritoneal cavity. The remaining 47 were secondary to inflamed and perforated organs within the abdomen; 35 followed rupture of the appendix, and 12 followed other catastrophes. It is my purpose to discuss briefly these three kinds of problems with particular emphasis on the primary type, of which one case of primary streptococcal peritonitis was treated recently. Also, it should be mentioned that the cases were treated by many different private practitioners, but the approach was remarkably constant.

DR. MERTZ

Primary Peritonitis

Lipshutz[1] in 1926 described pneumococcic or streptococcic peritonitis as the most terrible abdominal catastrophe of infancy and early childhood. He treated 23 cases in three years, 14 pneumococcal and 9 streptococcal, of which 18 received surgical drainage. All 23 patients died. During the next decade very little improvement can be demonstrated. In 1937, Leopold and Kaufman[2] of the Willard Parker Hospital, New York, reported 12 cases of streptococcal peritonitis with one recovery. In 1939 Ladd[3] advocated the combined use of surgery and specific therapy with sulfa drugs, which at the time dramatically reduced the mortality rate in his large series.

Although this condition has been described at all ages, it is commonly a disease of infants and children. The incidence is considered to be much reduced at present, and the paucity of literature in English in the last ten years supports this, although a number of articles in other languages were noted. However, of the 14 cases reported, 11 occurred after 1950. Ten or 72 per cent were aged five or younger, concurring with classical descriptions of the disease. The sex distribution was exactly one to one. Three (21 per cent) of the 14 died, two within 24 hours of admission to the hospital, and the third 33 days after surgical and massive antibiotic therapy was given, succumbing finally to the combined streptococcal and proteus infection.

Ladd and Gross (1950)[4] reported a mortality of 18 per cent (of 22 patients) surgically treated and 37 per cent (of 16 patients) medically treated in comparison. Both groups received liberal antibiotic therapy.

The causative agents of this disease were found by Ladd and Gross[5] prior to 1940 to be the streptococcus and pneumococcus (ratio two to one, favoring streptococcus), with a very small number caused by colon or unknown organisms. During

* Resident, Kauikeolani Children's Hospital.

[1] Lipshutz, B., and Lowenburg, H.: Pneumococcic and streptococcic peritonitis. Report of twenty-three cases in infants and children, JAMA 86:99 (Jan. 9) 1926.

[2] Leopold, J. S., and Kaufman, R. E.: Acute "primary" streptococcus peritonitis, J. Pediat. 10:45 (Jan.) 1937.

[3] Ladd, W. E., Botsford, T. W., and Curnen, E. C.: Primary peritonitis in infants and children. More effective treatment, JAMA 113:1455 (Oct. 14) 1939.

[4] Gross, R. E.: The surgery of infancy and childhood, W. B. Saunders Co., Philadelphia, 1953, p. 390.

[5] Ladd, W. E., and Gross, R. E.: Abdominal surgery of infancy and childhood, W. B. Saunders Company, Philadelphia, 1940, p. 189.

the subsequent ten years the ratio was nearly reversed, but the pneumococcus was often associated with nephrosis. In the present 14 cases, three were pneumococcal, two were caused by beta hemolytic streptococci, three were staphylococcal, and the remainder unknown. The mode of entry most widely accepted is via the blood stream, and of six patients on whom blood cultures were made, three contained the offending organism.

Table 1 shows the most common symptoms, physical and laboratory findings in the cases under discussion. Abdominal pain and vomiting were the most common symptoms. Diarrhea and antecedent upper respiratory infections occurred in less than half the cases. Abdominal distension of considerable degree was nearly always present, and fever of 103° or higher was noted in seven patients. A white blood count over 20,000 was found in only half the patients. Generally speaking, however, these patients were described as extremely ill, in contrast to those with appendiceal rupture.

TABLE 1.—*Symptoms, Physical and Laboratory Findings, and Related Factors in 14 Cases of Primary Peritonitis.*

Years cases were seen:	
1943-47	1
1948-52	6
1953-58	7
Ages:	
0-2 years	9
2-5 years	1
5-10 years	4
Temperature 103° or higher	7
WBC over 20,000	7
Abdominal pain and tenderness	9
Vomiting	8
Diarrhea	5
Respiratory infection	5
Bowel sounds decreased or absent	9
Abdominal distension	10
Causative organisms:	
Pneumococcus	3
Streptococcus	2
Staphylococcus	3
Proteus with streptococcus	1
Pus, but no growth	3
Unknown	3
Patients with nephrosis	1

The differential diagnosis of primary peritonitis essentially involves the secondary type, or pneumonia. High fever, diffuse abdominal tenderness, and much distension in a very young child suggests a primary etiology, while vomiting, pain, tenderness, and rebound tenderness localized in the right lower quadrant, with slight fever, in an older child, suggests appendicitis. Pneumonia can usually be excluded by x-ray and the absence of abdominal tenderness.

Seven patients received surgical exploration, and three appendectomy at the time of surgery. One of those having an appendectomy died. Of seven managed medically, two expired, the first two hours after admission, and the second within the first 24 hours. All received antibiotics, usually penicillin, but invariably sulfa or a broad spectrum drug as well when available. Although this series is of small size, the percentage of success of surgical versus medical therapy alone is almost identical with Gross's figures, and it seems to commend his surgical approach.[6] This entails a small surgical incision to obtain pus, and if streptococci or pneumococci are found, simple drainage of the pelvic area with a drain followed by vigorous postoperative support, gastric suction, and specific antibiotics.

The following is a case report of the most recent case of primary peritonitis at Kauikeolani Children's Hospital.

Case Report

A three-month-old boy was admitted with complaints of fever for three days, reaching 104° to 105° F., vomiting all feedings for 18 hours, fussiness for 12 hours and severe diarrhea and distension for six hours prior to admission. Physical examination revealed an extremely ill-appearing infant with a temperature of 105° F., moderate dehydration, and a distended, quiet, taut abdomen.

The initial white blood count was 10,700 with 34 neutrophiles, 49 stabs and 17 lymphocytes, with hemoglobin of 9.2, and hematocrit of 32 per cent. The urine was unremarkable. Admission electrolytes revealed CO_2 16 mEq, chlorides 98 mEq, potassium 3.9 mEq, sodium 122 mEq. Chest x-ray suggested a right upper lobe pneumonitis, and x-ray of the abdomen revealed a ground-glass appearance and a loss of the fat line.

Peritoneocentesis performed by the surgical consultant revealed thin odorless pus containing chains of gram-positive cocci which proved to be beta hemolytic streptococci. Blood cultures also grew this organism.

The surgeon elected to explore the abdomen and 300 cc of the same type of pus were removed. After exploration, the abdomen was drained through two lateral stab wounds. Two million units of aqueous penicillin were given daily for two weeks and chloramphenicol (Chloromycetin) one gram daily for one week. In addition, the patient received gastric suction, intravenous therapy, and vigorous general support as long as necessary. The patient developed a slight elevation of temperature on the fourth and eighth postoperative days, but was otherwise afebrile after surgery, and was discharged on the twenty-first day entirely well.

Secondary Peritonitis

Appendiceal rupture accounted for 35 of 47 cases in this category. In contrast to the primary cases, 27 of them were five years or older (77 per cent). The ratio of males to females was one to one. Among the 35 there was one death. This occurred ten hours after admission, the patient being moribund when first seen.

Abdominal pain, usually most marked in the right lower quadrant, occurred almost universally, and vomiting was almost as common. The admitting temperature was nearly always less than 103°

[6] Gross,[4] p. 388.

F. Of interest was the finding of a white blood count over 20,000 in 31 per cent, versus 50 per cent in the primary cases, suggesting this to be an important difference, but not so great as usually believed. In both types, 85 per cent have counts greater than 10,000 at first examination.

TABLE 2.—*Symptoms, Physical and Laboratory Findings and Related Factors in 35 Cases of Ruptured Appendix.*

Years cases seen:	
1943-47	13
1948-52	10
1953-58	12
Ages:	
0-2 years	3
2-5 years	5
5 or more years	27
Temperature 103° or higher	8
WBC over 20,000	11
Abdominal pain and tenderness	35
Vomiting	31
Abdominal distension	2

Thirty of 35 received primary appendectomy with recovery on the average in 13 days. Three had secondary appendectomy after initial drainage and did well. One case was treated medically without drainage, and surgery done one month later. The one fatal case received no surgical therapy in the short time before death. Table 2 summarizes these cases.

TABLE 3.—*Causes of Secondary Peritonitis in 12 Nonappendiceal Cases.*

LESIONS FOUND	NUMBER	REMARKS
Perforation, duodenal ulcer	1	Died at 13 days. Culture—Staphylococcus 81
Rupture Meckel's diverticulum	2	Child age two lived. Child one month died at two months of multiple complications and peritonitis
Perforations of colon and rectum	5	Died within one week
Volvulus with peritonitis	2	Died within one week
Adhesions with sterile peritonitis	1	Recovery

The 12 cases of secondary peritonitis of nonappendiceal origin represent a discouraging group with a high mortality. Table 3 summarizes the various causes noted. All except three were below three months of age, and seven were younger than one month on admission. Nine received specific surgical therapy for the lesion noted. Ten patients expired. All died within 12 days of admission with the one exception of an infant with congenital volvulus who lived six weeks. The two survivals were: (1) a two-year-old boy with a rupture of a Meckel's diverticulum (he received appendectomy and diverticulectomy) and (2) a seven-year-old boy with adhesions and obstruction, but a sterile peritonitis. Eleven of the 12 patients were males.

Conclusions

1. Primary peritonitis, though less common than formerly, still occurs and represents a serious problem. A combined surgical and medical approach is recommended.

2. Peritonitis related to appendiceal rupture is common, but should yield a satisfactory result.

3. Secondary peritonitis of nonappendiceal origin in young infants carries a very serious prognosis.

Summario in Interlingua

Primari peritonitis pneumococcal, streptococcal, o staphylococcal del infantia e pueritia ha cessate esser invariabilemente mortal. Drainage chirurgic insimul con therapias antibacterial ha reducite le mortalitate dramaticamente. Un caso del condition es reportate.

Peritonitis secundari del infantia e pueritia ha un prognose satis bon. Illo tende a occurrer in patientes de etates plus avantiate, a generalmente parlar, e le mortalitate es basse quando le causa es ruptura del appendice. Al Hospital Juvenil Kauikeolani, 35 tal casos ha essite vidite in le curso del passate 15 annos, con un sol morte. Durante le mesme periodo, 12 altere casos de origine non appendiceal esseva vidite, e in iste serie il habeva 10 mortes.

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Jaundice within 36 hours of birth may be due to ABO incompatibility, and it can lead to kernicterus!

Hemolytic Disease of the Newborn Due to ABO Incompatibility

S. H. WAXMAN, M.D.,* Honolulu

RH-INCOMPATIBLE hemolytic disease is uncommon in Hawaii because of the high Oriental population and the low incidence of Rh negative blood type among Orientals. ABO-incompatible hemolytic disease is as common in Hawaii as elsewhere. This review of hemolytic disease of the newborn due to ABO-incompatibility is presented with the hope that it will make this disease more understandable and more easily recognized.

ABO-incompatible hemolytic disease is caused by action of maternal anti-A or anti-B antibodies upon fetal erythrocytes of the corresponding (A or B) blood group. Physiologic jaundice and icterus praecox of heterospecific pregnancy may fall under the condition of this definition.

Heterospecific pregnancy, occurring in 25 per cent of all pregnancies, is any pregnancy in which an infant has an A or a B agglutinogen which the mother does not possess, and against which she may have antibodies.

Icterus neonatorum praecox was described by Halbrecht[1] in 1944 as a benign, neonatal jaundice commencing within 24 hours of birth, and unaccompanied by any blood changes apart from an occasional mild anemia. This, of course, closely resembles the definition of physiologic jaundice except for the time element. Physiologic jaundice usually occurs after 36 to 72 hours.

* Senior Resident in Pediatrics, Kauikeolani Children's Hospital.

[1] Halbrecht, I.: Role of hemo-agglutinins anti-A and anti-B in pathogenesis of jaundice of the newborn (Icterus Neonatorum Praecox), Am. J. Dis. Child. 68:248 (Oct.) 1944.

Incidence

Because of the lack of a clear-cut definition, the incidence of this disease has varied. At one time, it was felt to be a rare disease. In the last decade, however, it has been found much more often, and at present is considered by many to be more common than Rh incompatibility. With the higher incidence of Group O individuals (47 per cent) and Group A individuals (42 per cent) as compared to Rh negative individuals (14 per cent), ABO incompatibility should be more common.

DR. WAXMAN

Table 1 gives the incidences as found by various authors over the last few years and around the country. From this table, the incidence of the disease seems to be rising, probably due to better recognition. A number of authors have found that hemolytic disease due to ABO incompatibility is more common than hemolytic disease due to Rh incompatibility. Gunson[9] has reported four cases in which combined ABO and Rh incompatibility were found. Both factors played a role in the re-

TABLE 1.—*Incidence of ABO Hemolytic Disease.*

SOURCE	YEAR	NO. OF DELIVERIES	Rh	Rh RATIO	ABO	ABO RATIO
Ohio State U.[2] Health Center	1953		9		3	
	1954		20		4	
	1955		26		18	
Royal Vic. Mat.[3] Hosp. Montreal	1954		24	1:140	3	1:1000
	1955		27	1:120	11	1:300
Confederate[4] Memorial Med. Center	1955	4265			9	1:475
	1956	4511	7		16	1:350
	1957 (3 mos.)	1118	....		6	1:190
Turman, *et al.*[5]	1955	2672	....	.....	23	1:110
S. L. Leiken, *et al.*[6]	1957	1125				
	?	(230 heterospecific)			12	1:195 (69% of heterospecific)
Beth Israel[7] Boston	6 mos.					
	1953	1550	7	1:225	11	1:150
	1 year					
	1953	2624	14	1:180	19	1:130
St. Thomas[8] Hospital, Canada	1957	.....				
	1 year	1000	7	1:140	14	1:70 ((7% of heterospecific)

TABLE 2.—*Incidence of Primipara Showing ABO Hemolytic Disease.*

AUTHOR	NO. OF CASES	NO. OF 1ST BORN	PER CENT
Valentine[8]	14	3	22
Hsia and Gellis[7]	21	9	43
Zuelzer[10]	220	?	40-50

TABLE 3.—*ABO Types in Hemolytic Disease.*
Over 90% have O-Mother
About ⅔ of all Cases are O-Mother
A-Infant (O-A)

AUTHOR	CASES	O-A	O-B	OTHERS
Zuelzer[11]	44	34	9	1 (A-B)
Stern *et al.*[12]	34	27	7	.....
Shumway *et al.*[13]	10	7	2	1 (A-B)
Lowenstein *et al.*[3]	17	17	..	.....
Hsia and Gellis[7]	21	21	..	.....
Zuelzer[10] (enlarged series)	207	142	63	2 (A-B)

[2] Copeland, W. E., Vorys, N., and Ullery, J. C.: ABO incompatibility in the etiology of hemolytic disease of the newborn, Am. J. Obst. & Gynec. 73:1045 (May) 1957.

[3] Sabin, M., and Lowenstein, L.: Hemolytic disease due to ABO isoimmunization, Canad. M. A. J. 77:92 (July 15) 1957.

[4] Bodran, M. A., Jr., and Cotter, J. W.: ABO incompatibility, J. Louisiana State Med. Soc. 109:450 (Dec.) 1957. Copeland.[2] Sabin.[3] Hsia.[7]

[5] Turman, C., Vaughn, V. C., and Shelley, R. M.: Incidence of hemolytic disease of the newborn due to A or B incompatibility, Am. J. Obst. & Gynec. 71:885 (Apr.) 1956.

[6] Leiken, S. L., Rheingold, J. J., and Sotes, J. G.: Frequency of ABO erythroblastosis, Pediatrics 22:65 (July) 1958.

[7] Hsia, D. Y. Y., and Gellis, S. S.: Studies on erythroblastosis due to ABO incompatibility, Pediatrics, 13:503 (June) 1954.

[8] Valentine, G. H.: ABO incompatibility and hemolytic disease of the newborn, Arch. Dis. of Child. 33:185 (June) 1958.

[9] Gunson, H. H.: Combined Rh and AB hemolytic disease of the newborn, Am. J. Clin. Path. 27:35 (Jan.) 1957.

[10] Zuelzer, W. W., Cohen, F.: ABO hemolytic disease and heterospecific pregnancy, Pediatric Clinics of N. America: Pediatric Hematology, 405 (May) 1957.

[11] Zuelzer, W. W., and Kaplan, E.: ABO heterospecific pregnancy and hemolytic disease. IV. Pathologic variants, A.M.A. J. Dis. Child. 88:319 (Sept.) 1954.

[12] Stern, H., Davidsohm, J., and Budzitsky, A.: Neonatal serological diagnosis of hemolytic disease of the newborn caused by ABO incompatibility, J. Lab. and Clin. Med. 50:530 (Oct.) 1957.

[13] Shumway, C. N., Miller, G., and Young, L. E.: Hemolytic disease of the newborn due to anti-A and anti-B, Pediatrics 15:54 (Jan.) 1955.

sultant hemolytic disease. Two other facts with regard to incidence are of importance. First, there is a high incidence in first-born infants (Table 2). Secondly, in almost all reported cases, the mother was type O, while the infants were either type A or type B. Of these, about three-fourths were O-A incompatible, that it, the mother was type O and the infant type A.

Table 3 gives relative proportions of blood grouping in the cases of various authors.

Pathogenesis

Theoretically, the mother is of a blood group differing from that of the infant's and so can form antibodies against the infant's blood group. The infant's antigens (its blood group) stimulate the formation of maternal antibodies, which in the infant can cause hemolysis and hemolytic disease. This presupposes free placental transfer of both antigens and antibodies. If this were true, then all heterospecific pregnancies should result in hemolytic disease; that is, 25 per cent of all pregnancies should result in hemolytic disease. If we take the highest incidence reported, that of Valentine's,[8] seven per cent of all heterospecific pregnancies result in hemolytic disease. Thus, this concept is not true. Three factors may play a part in keeping this incidence low: 1. The antibodies found are not the iso-antibodies found normally, but are immune antibodies. 2. There is some type of placental barrier or protection. 3. There is some fetal protection.

Nature of the Antibodies

It is generally agreed that the antibodies are not the natural occurring or physiological antibodies.

Here, however, there is some controversy. The majority of authors[14] feel that the offending antibodies are the so-called immune or incomplete antibodies. These antibodies show one or more of the features listed in Table 4.

TABLE 4.—*Properties of Immune Antibodies.*

1. Thermal optimum of 37° C.
2. Greater resistance to heating at 70° C.
3. Higher conglutination titers in colloid media than saline media.
4. Positive Coombs in dilutions beyond end-point of titration in saline.
5. Resistance to neutralization by soluble A or B blood type substance.
6. Hemolysis and complement fixation *in vitro*.

Stern *et al.*,[12] in their series, found this type of immune agglutinin (or antibody) in all of their 34 cases. In 123 heterospecific pregnancies without evidence of hemolytic disease, they found only 14 cases containing these immune antibodies; this is 11 per cent of their cases. There are few series in which the antibodies (or agglutinins) were studied as intensively. But using the above criteria for immune antibodies, most authors,[14] in reporting their series, found that the antibodies exhibited some of these features and could be called immune.

Wiener[15] has postulated and cited some evidence for another factor that is shared by A and B blood types, but is absent in the O blood type. He has called this factor C. Since almost all ABO-incompatible pregnancies occur in type O mothers, this factor could be present in the fetus and would not be present in the mother. Thus, this factor could be the cause of the formation of the immune antibodies. This factor has not been isolated, nor are there any specific tests for it.

A third explanation hypothesizes that there is a cross-reactivity between the natural occurring anti-A and anti-B antibodies. The resultant antibody is the immune antibody. Zuelzer, Cohen, and Robinson[16] report a case in which a type O mother gave birth to twins, one having B blood type and the other A. The type B infant exhibited hemolytic disease. Their studies indicated that the hemolytic disease was caused by a specific anti-B antibody. They were able to demonstrate cross-reacting antibodies as well as anti-B and anti-A antibodies. They found that these cross-reacting antibodies had only a partial or loose affinity for the cells, and did not have much to do with the hemolytic disease. They felt that this proved only that type O serum contains variable complexes of anti-A and anti-B antibodies, composed of multiple fractions with different partial specificities.

One other factor about antibodies is of interest. The maternal antibody in O-A incompatible hemolytic disease tends to have an A_1 specificity. This fact was first reported by Witebsky.[17] Zuelzer[11] found that this was true in his cases. Zuelzer showed that in 142 type A infants, 82 were subgroup A_1 at birth. Of the 54 type A_2 at birth, 14 were tested at a later date and found to be type A_1. Besides the maternal specificity, it is also known that A_1 cells have a greater reactivity. Zuelzer therefore feels that probably only A_1 and B infants are susceptible to hemolytic disease due to ABO incompatibility.

Placental Barrier

Some authors have postulated that the placenta forms a barrier for the transfer of antibodies. This could account for the variable incidence of hemolytic disease due to ABO incompatibility. This theory, however, does not seem to hold true. Zuelzer and Kaplan[18] in their studies, tested both maternal and infant sera of homospecific and heterospecific pregnancies for antibody titers. In homospecific pregnancies, they found that anti-A and anti-B antibodies, both natural and immune, were present in the vast majority of cord sera, and had thus crossed the placental barrier. The height of the fetal titers was limited by the maternal titers. In heterospecific pregnancies, they found that the antibodies antagonistic to the fetal blood types were nearly always absent from the cord serum.

Stern *et al.*[12] and Polayes *et al.*[19] had similar results. Stern found anti-A antibodies in 95 per cent of his cord sera, when present in the maternal serum. Anti-B antibodies were found in 79 per cent of the cord sera. This was in normal infants and mothers of both heterospecific and homospecific pregnancies. He also found in his heterospecific pregnancies that the antagonistic antibody was nearly always absent. This latter observation has led to a good presumptive test for ABO incompatible hemolytic disease.

In summary, the placenta seems to be freely permeable to all antibodies, immune or physiological.

Fetal Protective Mechanism

The work of Zuelzer,[18] Stern,[12] and Polayes[19] indicates that the fetus has protective mechanisms by which it is able to neutralize maternal antibodies. It is known that infants can be secretors or

[14] Rosenfeld, R. E.: A-B hemolytic disease of the newborn. Analysis of 1480 cord bloods. Blood X:17 (Jan.) 1955.
Levine, P., Robinson, E., Stroup, M., McGee, R., and Bushnell, L. N.: A summary of atypical antibodies, rare genotypes and ABO hemolytic disease encountered in a one year survey. Blood XI:1097 (Dec.) 1956.

[15] Wiener, A. S: The blood factor C of the ABO system. Ann. Eugenics 18:1 (June) 1953.

[16] Zuelzer, W. W., Cohen, F., and Robinson, A. R.: Specific and cross-reacting antibodies in ABO heterospecific twin pregnancy. Blood XII:883 (Oct.) 1957.

[17] Witebsky, E., and Engasser, L. M.: Blood groups and subgroups of the newborn. I. The A factor of the newborn. J. Immunol. 61:171, 1949.

[18] Zuelzer, W. W., and Kaplan, E.: ABO heterospecific pregnancy and hemolytic disease. II. Patterns of A and B isoantibodies in cord blood of normal infants. A.M.A. J. Dis. Child. 88:179 (Aug.) 1954.

[19] Polayes, S. A., Lederer, M., and Weiner, A. S.: Studies in isohemagglutination. J. Immunol. 17:545 (Dec.) 1929.

nonsecretors; that is, they may or may not have appreciable amounts of type A or B substance in their plasma, saliva, gastric juice, and tissue fluids. It was thought that this substance could be the neutralizing substance in the fetus. Thus, nonsecretors should be the ones suffering from hemolytic disease, whereas secretors should be immune. However, both secretors and nonsecretors have been found to have hemolytic disease of the newborn due to ABO incompatibility. Zuelzer,[20] in his study of heterospecific normal pregnancies, showed that both nonsecretors and secretors had an absence of the antagonistic antibodies. Further study revealed that both secretors and nonsecretors have potent blood type substances in their tissue cells. These probably play an important role in neutralizing the offending maternal antibodies and sparing the erythrocytes.

The relatively low reactivity of fetal erythrocytes for A or B antibodies could be another fetal protective device. This has been suggested by the fact that the hemolytic activity, agglutinin titers, and affinity for anti-A and anti-B antibodies are weaker for fetal cells than for adult cells. Also, Zuelzer[11] and Witebsky[17] both showed that the development of A antigen is not always complete at birth. Since the maternal antibody shows an A_1 specificity, this would offer some fetal protection.

The most important fetal protective mechanism may be the one that differentiates the so-called normal ABO-heterospecific pregnancies from the hemolytic disease of the newborn due to ABO incompatibility. This is the ability of the liver to handle and excrete bilirubin. If this ability is normal, then the hemolysis due to incompatibility may go on without any outward manifestations of distress. If this ability is poor, then even a normal load of bilirubin can cause hemolytic disease of the newborn as manifested by jaundice, etc. At present this ability has not been fully elucidated. It is thought to be a function of specific enzymes which may be lacking or not fully developed in the newborn liver.

In summary, the fetal protective mechanisms seem to be the most important. All fetuses have potent antigens in their tissue cells which can neutralize offending antibodies. Fetal erythrocytes are of low reactivity and type A is not fully differentiated into A_1 and A_2 at birth. Finally, the liver plays a very important role.

Diagnosis

Jaundice in the first 24 to 36 hours should always be considered pathologic. This is especially true in hemolytic disease due to ABO incompatibility. Jaundice is usually the presenting, and in most cases the only, clinical manifestation. Pallor due to anemia can occur, but is not common. The clinical features of erythroblastosis, even to the delivery of a hydropic fetus, have been reported but are uncommon. The liver and spleen may be slightly or moderately enlarged. Purpura and bleeding are almost never encountered. With this bare clinical picture, how do we make the diagnosis? The clinical picture, an awareness of the disease, and the criteria listed in Table 5 should be helpful.

TABLE 5.—*Diagnosis of ABO Hemolytic Disease.*

1. Jaundice within first 24-36 hours.
2. First born in 40-50 per cent.
3. Mother type O and infant type A or B.
4. Exclusion of other hemolytic factors such as Kell, Duffy, Dias, etc.
5. Exclusion of sepsis, toxoplasmosis, lues, etc.
6. Mild or no anemia in presence of jaundice.
7. Spherocytosis.
8. Mild to moderate reticulocytosis.
9. Increased osmotic and mechanical fragility.
10. Negative or only mildly positive Coombs' test.
11. Immune antibodies demonstrated in maternal serum.
12. Free antibodies in infants serum capable of agglutinating erythrocytes of the infant's blood type.

Not all these criteria need be met. Hsia and Gellis[7] feel that clinical jaundice within 24 hours, the presence of a major blood group incompatibility, and a negative or only mildly positive Coombs's test, are sufficient for a diagnosis.

Tests for the demonstration of the latter two criteria in Table 5 are cumbersome and time-consuming. The literature is full of variants and different tests done to better facilitate these findings. At the Blood Bank of Hawaii the fetal blood is tested. The fetal serum is tested against type A, B, and O cells for the presence of antibodies. The fetal cells are eluted and the eluates tested against type A, B, and O cells for antibodies. In both tests, if antibodies A and B are found, the infant is considered to have ABO-incompatible disease. The Coombs's test, as mentioned above, is rarely positive (30 to 60 per cent), and if it is positive, it is usually only mildly so.

In Rh-incompatible pregnancies, the maternal blood can be tested for rising titers so that the obstetrician and pediatrician can be prepared for an infant with hemolytic disease. This cannot be done in ABO-incompatible pregnancies. Zuelzer[22] found that the majority of heterospecific pregnancies exhibit a stationary titer throughout pregnancy. Only a relatively few will show a rise in titer, but this usually occurs in the postpartum

[20] Zuelzer, W. W., and Kaplan, E.: ABO heterospecific pregnancy and hemolytic disease. III. Hematological findings and erythrocyte survival in normal infants, A.M.A. J. Dis. Child. 88:907 (Sept.) 1954.

[21] Arnold, D. P., Witebsky, E., Selkirk, G. H., and Aford, K. N.: Clinical and serological experiences in treating hemolytic disease of the newborn, J. Pediat. 46:506 (May) 1955.

[22] Zuelzer, W. W., and Kaplan, E.: ABO heterospecific pregnancy and hemolytic disease. I. Patterns of maternal A and B isoantibodies in unselected pregnancies, A.M.A. J. Dis. Child. 88:158 (Aug.) 1954.

period. This response does not correlate with hemolytic disease due to ABO incompatibility.

Therapy

The benignity of hemolytic disease of the newborn due to ABO incompatibility has been stressed too frequently. Kernicterus may occur in ABO incompatibility, and most authorities feel that the level of bilirubin in all these patients should be followed closely and exchange transfusions done. Zuelzer[10] uses the following criteria for exchange transfusions.

TABLE 6.—*Therapy.*

1. Watchful observation with frequent bilirubin determinations.
2. Supportive therapy.
3. Exchange transfusions.[10]
   a. Full-term infants
      Bilirubin greater than 10 mg per cent in 1st 24 hours.
      Bilirubin greater than 14 mg per cent in 2nd 24 hours.
      Bilirubin greater than 17 mg per cent in 3rd 24 hours.
   b. Prematures
      Slightly lower levels than "a".
4. Simple transfusions for anemia.

In full-term infants, if the bilirubin is more than 10 mg per cent in the first 24 hours, more than 14 mg per cent in the second 24 hours, and more than 17 mg per cent in the third 24 hours, an exchange transfusion should be done. If the bilirubin levels are less than those mentioned, the patient's condition and bilirubin should be followed every four to eight hours and an exchange transfusion done if it is found that the patient's condition is deteriorating or the bilirubin is increasing rapidly. In prematurely born infants, slightly lower levels are used as criteria for exchange transfusion. Second, third, and even fourth exchanges have been reported although these are not common.

Hyman and Sturgeon,[23] and Hsia and Gellis[7] use a slightly broader criterion for exchange transfusion. They advocate exchange transfusion in any infant whose bilirubin is 20 mg per cent, or rapidly approaching this level, and who is less than 72 hours old. In prematurely born infants 96 hours is the time limit used.

Sabin and Lowenstein[3] use 10 mg per cent bilirubin in the first 24 hours as a critical level. Also a hemoglobin of 15 gm per cent is critical. An exchange transfusion will depend on the clinical condition of the child and these levels.

Most authors [24] feel that the type of blood used should be fresh blood (less than three days old) of type O, Rh type the same as the infant. Table 6 summarizes the therapy of ABO-hemolytic disease. Most reported series show exchange rates of 40 to 70 per cent of ABO cases.[25]

Supportive therapy, such as oxygen, antibiotics, etc., should be given. Anemia with a low bilirubin can be corrected by simple blood transfusion. ACTH and cortisone have been used but no convincing reports as to their efficacy have been published.

Prognosis

Prognosis is usually good. Kernicterus is virtually the only danger. The incidence of kernicterus is hard to evaluate. Most reports of kernicterus are single case reports. Dunn[26] reports a pair of twins with hemolytic disease due to ABO incompatibility, both of which developed kernicterus. Zuelzer[16] reports kernicterus as 10 to 15 per cent in a series of severe cases of hemolytic disease. Copeland *et al.*[2] found two patients with kernicterus out of a total of 29 patients. This is about six per cent. Many other authors have found no kernicterus at all in their series. In untreated cases, kernicterus appears to occur in 5 to 15 per cent of the cases. In treated cases, two per cent develop kernicterus. The kernicterus seems to be related to the height of the serum bilirubin.

In the long-term follow-up care most authors have found that these children do extremely well. A few of them will develop anemia in the neonatal period, but this can usually be treated medically and does not need transfusion.

Summary

A review of ABO-hemolytic disease is presented. The pathogenesis of this disease is as yet not fully understood. The incidence is steadily rising, probably due to better recognition, and many authors feel that this disease is much commoner than Rh hemolytic disease. The diagnosis is discussed with stress being placed on the fact that jaundice occurring in the first 24 to 36 hours is usually pathological. Treatment is similar to that used in any of our hemolytic diseases of the newborn with exchange transfusion being the main modality. Prognosis is good but kernicterus can and does occur.

Summario in Interlingua

Jalnessa neonatal comenciante intra 36 horas post nato pote esser le effecto de morbo hemolytic a incompatibilitate ABO o de incompatibilitate Rh, si le typo sanguinee del infante es A_1 o B e illo del matre O. Le pathogenese del morbo es non ancora completemente comprendite. Le incidentia cresce in tanto que le recognition del condition es meliorate. Ben que le prognose es bon, le condition pote ducer a ictero nuclear (kernicterus). Le principal methodo therapeutic es transfusiones de excambio.

[23] Hyman, C. B., and Sturgeon, P.: Management of erythroblastosis foetalis and ABO hemolytic disease, West. J. Surg., Obst. and Gynec. 65:384 (Nov.-Dec.) 1957.
[24] Sabin,[3] Hsia,[7] Valentine.[8]
[25] Copeland,[2] Sabin,[3] Bodran,[4] Hsia.[7]
[26] Dunn, H. G.: Hemolytic disease of the newborn due to ABO incompatibility, A.M.A. J. Dis. Child. 85:655 (June) 1953.

The premature infant's immaturity is not merely anatomical—
it is physiological as well, and we must not forget this.

Some Aspects of Physiology in the Premature Infant

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LIFE is precarious for the premature infant and the tragedy of it is that we are often unable to give much help. When death supervenes, we attribute it to prematurity when, in fact, the cause of death is too often not known. Pathological processes can only be understood when compared to the normal physiology. Therapy also must be instituted with full knowledge of what constitutes the normal. It is not possible to extrapolate knowledge of the adult to the newborn and especially to the premature and come up with the right answers.

DR. HAGINO

In 1955, in the United States, perinatal mortality was designated as the fourth largest cause of death during the whole life span. Of this group prematurity was by far the diagnosis most often submitted as the cause of death.[1] If one considers seventy years as the normal life span, then the total number of potential human years lost is staggering.

Criteria of Immaturity

A diagnosis of immaturity is an arbitrary diagnosis. The single criterion most often used is a weight of 5½ pounds (2,500 gm) or less. Other objective criteria that have been used are crown-heel length, gestational age, head measurements, chest circumference, ossification centers, and combinations of these.[2]

E.D.C. or expected date of confinement depends upon exact information as to the date of onset of the last menstrual period. Naegele's rule of adding 7 days to the date of the first day of the last menstruation and counting back 3 months to obtain the E.D.C. is subject to much error. When it is used, however, an infant born two or three weeks before term is generally regarded as premature.

The diagnosis of prematurity based on length at birth is not reliable. Observers are not agreed as to the critical length. In general, a crown-heel length between 35 and 47 cm (16 to 19 inches) is considered to be adequate for a viable premature.

It is generally agreed that a head circumference of less than 33 cm probably indicates prematurity. Measurement of the thorax at the nipple line of less than 30 cm has also been mentioned. According to Potter, a difference between head and thorax measurements of more than 3 cm probably indicates prematurity. Roentgenographic demonstration of certain centers of ossification such as the proximal tibial epiphysis, the cuboid, and the capitate have been investigated also as an indication of maturity. However, this technique also is invalidated because of sex and race differences in the ages of the appearance of the centers. For an adequate understanding of the physiology of prematurity, it is important to realize that during prenatal life, growth of all parts of the body does not proceed at the same rate. Hence, although the fetus is considered to be viable after 28 weeks of gestation, this does not mean that all organs are at the same stage of development.

At the end of two months almost all the internal organs are well laid down. The relationship of

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[1] Eliot, M. M.: Deaths around birth—the national score, J.A.M.A., 167:945 (June 21) 1958.
[2] Dunham, E. C.: Premature Infants. A Manual for Physicians. 2d edition, New York: Hoeber-Harper, 1955.

gestational age, weight, height, and clinical development are given briefly in the following table.[3]

MONTH OF GESTATION	WEIGHT (GRAMS)	HEIGHT (CM)	DEVELOPMENT
3rd	14	8.5	Recognizable human, nails formed, sex distinguishable
4th	100	15.5	Fetal movements felt by mother
5th	300	23.5	Hair formed on head and body
6th	675	31.0	Lean but proportion better, eyebrows and lashes present
7th	1,075	36.0	Skin red and wrinkled, eyelids reopen
8th	1,700	40.5	Testes enter scrotum
9th	2,400	45.0	Skin smoother, limbs more rounded, nails project
10th	3,333	50.0	Progressive fat accumulation, downy hair coat beginning to shed

Respiratory System

Respiratory dysfunction occupies a major part of the physician's concern for the premature infant. The most important limiting factor will be the degree of maturation of the fetal lung. If the pulmonary tissue of a premature infant is not sufficiently developed for performing gaseous exchange, then nothing in the way of expanding the lung or of providing oxygen will be of any help.

Unlike most other vital organs, the lung has no apparent function during intrauterine life. It must complete its development and be prepared to function as soon as the baby is born. It is not unlike the parachute which must function as soon as the ripcord is pulled. No alveolar tissue is present until the fifth month and the rudimentary bronchioles are lined only with cuboidal epithelium which cannot absorb oxygen. During the sixth month, respiratory epithelium develops and by the seventh month, there is enough to maintain life. However, development continues, so that the more mature the fetus, the greater the number of functioning alveoli. The immature lung is also deficient in elastic tissue so that proper expansion is interfered with and collapse occurs readily.[4]

As mentioned above, the fetal lung is passive. Any respiration in the sense of gaseous exchange takes place through the placental circulation. After the seventh month, the lungs are capable of carrying on respiration if breathing can be established. Actually, the mechanisms of breathing have been going on in intrauterine life even before viability, the only difference being that amniotic fluid instead of air entered the lungs.[5] The movements of the thorax and lungs are minimal so that they are not distinguished clinically. The question of whether these respiratory movements are regular or initiated only in response to certain stimuli such as anoxia, increased carbon dioxide tension, or mechanical irritation is still controversial.

The amniotic fluid which fills the fetal lung begins to escape as the amniotic sac breaks during labor. The excess fluids in the baby's lungs, gastrointestinal tract and body tissues are squeezed out by powerful uterine contractions. These same contractions act as stimuli to arouse the baby to a high degree of muscular activity. The practice of making a routine episiotomy and using forceps to protect the oncoming head in delivering premature infants has been questioned in the light of the high incidence of prematures who have respiratory difficulties as compared to those who develop intracranial bleeding.[6]

With delivery of the head the baby must make the transition from an aquatic to an air environment. Thus, the stage is set for the initiation of the first breath. Work done by Barcroft indicates that ordinarily respiration is probably initiated by sensory stimuli until an emergency situation arises, when the chemical stimuli become important.[7] Even though the respiratory center responds adequately to stimuli and there is a normal thoracic cage containing normal lung structures with open airways, certain mechanical factors have to be overcome for adequate respiration.

Inspiration is opposed by the elastic resistance of the lung tissue, surface tension, airflow resistance in the respiratory tract, and tissue viscous resistance.[8] The latter is the least of the factors and refers to the resistance caused by friction between molecules in moving tissue and is related to the velocity. Thus it differs from elastic resistance, which is a static factor related to the degree of expansion. During inspiration, both factors are working but at the end of inspiration, only the elasticity of the lungs needs to be balanced. During expiration the mechanical factors involved are the elastic recoil of lung tissue, surface tension, and the thoracic cage. They combine to help overcome airflow resistance and tissue viscous resistance. Among other factors which hinder respiration in the immature infant is the tendency of the larynx and trachea to collapse because of softness and the inability of the chest cage to withstand the negative pressures developed during inspiration.

Surface tension is probably the most inhibitory

[3] Arey, L. B.: Developmental Anatomy. A textbook and laboratory manual of embryology. 5th edition. Philadelphia: Saunders, 1946.
[4] Cole, W. C. C.: Resuscitation of the newborn premature infant. Ped. Cl. of North America (Aug.) 1954, p. 527.
[5] Davis, M. E., Potter, E. L.: Intrauterine respiration of human fetus. J.A.M.A. 131:1194 (Aug. 10) 1946.
[6] Barnes, A. C.: Physiology of prematurity. Josiah Macy, Jr. Foundation, 1957, p. 115.
[7] Barcroft, J.: Researches on prenatal life. Springfield: Charles C. Thomas, 1948.
[8] Karlberg, P. J. E.: Breathing and its control in premature infants. Physiology of prematurity. Josiah Macy, Jr. Foundation, 1957.

mechanical factor concerned with the first breath.[9] This is because of the air-fluid interface associated with the first breath. This force tends to contract the interface to the smallest possible area, which for any given volume is represented by a sphere. In the same way, when air is introduced into the newborn lung for the first time, it does not form a number of small bubbles which are distributed to the numerous alveoli but tends to form the fewest and largest bubbles in the lung. The size of bubble is limited by the architecture of the lung, so that as more air enters the lung more alveoli are expanded, requiring more pressure. Also, since the premature lung has relatively more bronchiolar capacity than alveolar, when some air is removed from the lungs, the remaining air is accommodated in the bronchi and the alveoli tend to collapse.[10]

The full-term infant is capable of producing enough pressure to initiate respirations by virtue of the high negative pressure resulting from expansion of the thorax, so that failure to breathe immediately after birth should make one think of abnormalities such as obstructed airway, damaged respiratory center, or severe anoxia. In the premature infant other factors as mentioned previously have to be considered. Compliance of the lung is a term used to designate the ratio of change in volume to change in pressure. It is variable with birth weight; i.e., the smaller the birth weight, the smaller the compliance. This means that more pressure is needed to expand the immature lung in the face of lung tissue which is more susceptible to rupture than the lung of the full-term infant. Wilson and Farber give a figure of 25 cm as the least water pressure on the trachea necessary to expand the lung in the full-term infant.[11] The use of positive pressure can be dangerous, as alveolar rupture and subsequent emphysema can occur with pressures of 40 cm of water for even short periods. Day advocates the use of short blasts of pressures up to and exceeding 40 cm for fractions of a second at frequent intervals.[12] They feel the shortened intervals of pressure to be less hazardous.

The respiratory rate of newborns is around 35 per minute. This holds true for premature infants down to 2,000 grams but for weights less than this higher values of about 40 to 50 per minute are found. Tidal volumes are about 15 to 20 ml and show good correlation with birth weights in weight groups above 2 kg but below 2 kg the tidal volume drops disproportionately. The minute volume is about 500 ml, the alveolar ventilation about 355 ml, and the functional dead space 5 ml.[13]

Once breathing has been established, the problem now arises of maintaining regular respirations. Irregular respirations and periodic (Cheyne-Stokes) breathing are considered by Clement Smith to be of frequent occurrence in the premature infant.[14] The question which occurs with the apneic spell is whether it is a benign episode or a forerunner of severe respiratory distress. Blystad in his determinations of blood gases in prematures with and without apneic spells found that infants without apneic spells; i.e., premature infants without any clinical evidence of respiratory difficulty, had no indication of respiratory insufficiency.[15]

Excluding cases of apnea due to hyaline membrane disease, he found that premature infants who had apnea in the course of the first four days of life had values similar to those seen in infants without any respiratory complications. In those cases who later developed apnea, respiratory acidosis and a tendency to moderate hypoxemia was demonstrated. These changes were also found during periods of effortless respirations. He felt that pulmonary changes as well as a central depression of the respiration were involved in the pathogenesis of this respiratory acidosis. While periodic (Cheyne-Stokes) breathing was often observed in infants where analysis showed a respiratory acidosis, he did not feel that low values for CO_2 in the blood constituted a precondition for the occurrence of this type of breathing in the premature infant.

In the same type of blood gas studies done on 73 prematures on the first day of life using heel puncture technique and ultramicro methods, Pincus and co-workers obtained the following values:[16]

15% had metabolic acidosis
29% had respiratory acidosis
38% had respiratory alkalosis
10% had a mixture of metabolic acidosis and respiratory alkalosis
4% were normal
3% were unclassified

After the first day of life, Reardon *et al.* found that 67 per cent had an uncompensated metabolic acidosis manifested by a lowered pH and total CO_2 content with a normal pCO_2. In ten per cent there was a mild respiratory acidosis and in seven

[9] Gruenwald, P.: Surface tension as factor in resistance of neonatal lungs to airation, Am. J. Obst. and Gynec. 53:996 (June) 1947.

[10] Levine, S. Z., Cook, C. D., Gruenwald, P., Silverman, W. A.: Respiratory difficulties of newborn infants. A panel discussion, N.Y. St. J. of Med. 58:372 (Feb.) 1958.

[11] Wilson, I. L., Farber, S.: Pathogenesis of atelectasis of the newborn, Am. J. Dis. Child. 46:572 (Sept.) 1933.

[12] Day, R., Goodfellow, A. H., Apgar, V., Beck, G. J.: Pressure-time relations in the safe correction of atelectasis in animal lungs, Pediatrics 10:593 (Nov.) 1952.

[13] Cook, C. D., Cherry, R. B., O'Brien, D., Karlberg, P., Smith, C. A.: Studies of respiratory physiology in the newborn infant. Part I. Observations on normal prematures and full-term infants, J. Clin. Invest. 34:975 (July) 1955.

[14] Smith, C. A.: Physiology of the Newborn, 2d edition, Springfield: Charles C. Thomas, 1951.

[15] Blystad, W.: Blood gas determinations on premature infants. Part 3. Investigations on premature infants with recurrent attacks of apnea, Acta Paed. 45:211, 1956.

[16] Pincus, J. B., Gittleman, I. F., Saito, M., Sobel, A. E.: A study of plasma values of sodium, potassium, chloride, carbon dioxide, carbon dioxide tension, sugar, urea and protein base binding power, pH and hematocrit on the first day of life, Pediatrics 18:39 (July) 1956.

per cent there was respiratory alkalosis. Thirteen per cent had normal acid-base balance and three per cent were unclassified.[17]

Since laboratory data tend to be controversial, the clinical studies done by Miller in relating birth weights and respiratory rates as a guide to survival and need for oxygen is of interest.[18] He proposed that oxygen therapy can be withheld with some confidence in cases of infants with birth weights from 1,001 to 1,750 grams provided the infants meet the following criteria:

1. Free from cyanosis within a few minutes of birth.
2. Spontaneous sustained respirations initiated within two minutes of birth.
3. Attain a respiratory rate of 40 per minute or more during the first hour.
4. Do not have significant increase in respiratory rate after the first hour.

He also found that when there was either initial apnea or bradycardia, 50 per cent died despite oxygen therapy. When there was combined initial bradycardia and apnea the prognosis was very poor indeed. He found, in those cases who developed later increase in rates of respiration during the first two days, that they sometimes showed initial improvement, then developed sudden apnea and bradycardia. Despite clinical improvement, the tidal volumes in these cases were reduced and remained so for a week or more.

Much work is being done in correlating rates of respiration to prognosis. Such being the case it should be noted that rates of respiration vary with positioning of the infant. Kravitz *et al.*, in observing 119 prematures and 49 full-term infants, found that there was a significant increase in respiratory rate in the prone position.[19] This was especially true in the lower weight groups of less than three days' age. If birth rate was more than 1,750 grams, then the rate was only slightly increased.

It can be seen that the physiology of respiration in the premature infant is complex and very labile. The diagnosis and treatment of any respiratory dysfunction is thus largely dependent on clinical acumen based on knowledge of normal physiology in the newborn.

Hematological Aspects

The blood of the premature infant is not unlike that of the full-term baby except in a few respects. They manifest a larger number of immature cells and have a striking tendency to develop an anemia after the first few months.

The immaturity of cells is seen in all cell types. Nucleated red cells are invariably present and reticulocytes may constitute eight to ten per cent of the total red cells. Variations in size and staining may not be marked but are always seen. The white cell series presents a similar picture, with immature cells which may persist for several months.

Almost all premature infants develop an anemia, which may be severe. Even though the cord hemoglobin may be equivalent to that of a full-term baby, the premature usually develops an abnormally low hemoglobin during the second month. This early anemia often improves from about four months, but is frequently followed by a second phase of anemia. This late phase is characterized by a hypochromia of red blood cells and responds to iron therapy, like the hypochromic anemia common in the other infants after six months.

The cause of the early anemia is not clearly understood but several mechanisms have been suggested:

1. Abnormally rapid red cell destruction.
2. Body growth outstripping normal erythropoiesis.
3. Functional immaturity of the bone marrow.

Gairdner studied 31 infants whose birth weights were less than 1,620 grams.[20] In order to elucidate which of the aforementioned theories caused the anemia of prematurity, he undertook several experimental studies. To see if there was actually a higher rate of destruction of red cells he examined the rate of decline of total hemoglobin during the first few weeks of life in all his infants. From the work of Mollison, it was known that among the red cell population of full-term infants there was a certain small proportion having a life span shorter than 120 days.[21] Hence, it becomes likely that a premature must also have such cells dating from an early period of fetal life. From his studies Gairdner concluded that the red blood cells of the premature infant did not break down at an abnormally high rate. Schulman *et al.* also calculated the rates of decline of total red cell mass and found that although the mean red cell life was slightly shorter than normal, it was not sufficient to account for the production of anemia.

The influence of body growth on the anemia of prematurity was studied in like manner. Premature infants have a weight gain of 1.5 to 2 per cent per day when they reach a weight of about four pounds (1,800 grams). In order to maintain a constant level of 11 gm/100 ml with a growth rate of two per cent per day the infant would have to produce 0.14 gm of hemoglobin per kg body weight per day over and above the depleted red

[17] Reardon, H. S., Graham, B. D., Wilson, J. L., Baumann, M. L., Tsao, M. U., Murayama, M.: Studies of acid-base equilibrium in premature infants. Pediatrics 6:753 (Oct.) 1950.

[18] Miller, H. C.: Studies of respiratory insufficiency in newborn infants, Part 3. Respiratory rates and birth weights of infants as guides to their survival and need for oxygen therapy, Pediatrics 22:432 (Sept.) 1958.

[19] Kravitz, H., Elegant, L., Block, B., Babakitis, M., Lundean, E.: The effect of position on the respiratory rate of premature and mature newborn infants, Pediatrics 22:432 (Sept.) 1958.

[20] Gairdner, D., Marks, J., Roscoe, J. D.: Blood formation in infancy. Part 4. The early anemia of prematurity, Archives Dis. Child. 30:203 (June) 1955.

[21] Mollison, P. L.: Blood Transfusion in Clinical Medicine. Springfield: Charles C. Thomas, 1951.

cells. To maintain the hemoglobin concentration at a somewhat lower level of 8 gm/100 ml would require the daily formation of 0.10 gm per kg or only 70 per cent as much. This means that the lower the hemoglobin concentration in the blood, the lower the amount of erythropoiesis needed to maintain this concentration during growth. It follows, therefore, that if erythropoiesis be insufficient to maintain a normal concentration, the concentration will continue to a level where hemoglobin requirements become balanced by hemoglobin formation.

Now if rapid growth were the main factor responsible for the early anemia of prematurity, then rate of growth should determine the degree of anemia. No such correlation could be found; however, it can be postulated that rapid growth is an important factor in prolonging the anemia, once it is produced.

The erythropoietic activity, as gauged by the narrow erythroid count, is high at birth, but falls rapidly to a very low level which persists for about two months, by which time the hemoglobin has usually fallen to about 11 gm per 100 ml, then slowly starts to rise. Normal full-term infants have the same picture.

To summarize the mechanism of early anemia of prematurity, then, the following facts are reviewed:

Suppression of erythropoiesis occurs shortly after birth in the premature as well as in the normal infant. As a result the hemoglobin concentration falls, due to a breakdown of red cells at an approximately normal rate. At a level of 11 to 12 gm, erythropoiesis is stimulated, but may not produce an appreciable effect on the blood for about two weeks, during which time the infant's hemoglobin continues to fall. This rate of fall is quite a bit faster in the premature infant because of his high rate of growth. His high rate of growth also makes correction of the anemia inevitably slow.

Iron stores in the developing fetus are derived entirely from its maternal supply of iron across the placental membranes. It was once believed that the greater part of this iron was laid down in the last months of pregnancy but reinvestigation by Widdowson and Spray indicates that the concentration of iron in the fetus remains constant throughout intrauterine life in amounts of approximately 7.5 mg per 100 gm of fetal weight.[22] The apparent quantitative increase in fetal iron during the last trimester of pregnancy is entirely due to its greatly increased growth during this period. This transfer of iron is reflected by an increased absorption of iron by the mother. There is evidence that the transplacentally acquired iron which is not present in the hemoglobin at the time of birth is used for hemoglobin synthesis later. Thus, the iron available for the formation of hemoglobin is not present in the circulating hemoglobin at birth. Severely anemic mothers seem to provide the infants with less iron than do normal mothers, but the physiologic variations in hemoglobin concentration in pregnant women do not indicate an inadequate transfer of iron to the fetus.

Anemia in the infant cannot be prevented by treating the mother. Some clinics routinely give all premature infants blood transfusions during the first few weeks of life. This procedure apparently helps the anemia but is only temporary, is not necessary in many cases, and presents some difficulty. Merritt and Davidson obtained good results by giving premature infants iron in the second week of life.[23] Many workers have added this as a routine treatment for prematures. Wolff gives an initial dosage at the time of discharge of 16 mg elemental iron per day.[24] The dosage is subsequently changed to 5 to 10 mg/kg/day as the infant grows. If dietary sources of iron are adequate after six months of age then therapeutic iron is discontinued.

Blackfan and Diamond, and Schulman *et al.* felt that iron was unnecessary before the third or fourth month.[25] The purpose of giving iron to premature infants is usually to prevent iron deficiency from developing after the first trimester; it is usually given about the sixth week or later. Gairdner feels that iron given early mitigates the anemia to a small but appreciable degree. He feels that there may be a stimulant effect upon hemoglobin synthesis even in the absence of iron deficiency.

In the well developed fetus active red cell production occurs not only in the red marrow of all the bones, but also in islands of erythrogenic tissue found in the liver and the spleen and to a lesser extent in other lymphoid tissue of the body. At term there is still some erythropoiesis going on in the spleen and less frequently in the liver, but after a few months the bone marrow assumes the entire burden. In the premature, therefore, extramedullary erythropoiesis must be a greater factor than in full-term infants.

Fowler studied the liver in 327 prematures, 205 of whom died within the first 72 hours, and found that red cell production in the liver varied inversely as the birth weight and that it fell off

[22] Widdowson, E. M., Sray, C. M.: Chemical development in utero, Arch. Dis. Child. 127:205 (Jan.) 1951.

[23] Merritt, K. K., Davidson, L. T.: The blood during the first year of life. Part 2. The anemia of prematurity, Am. J. Dis. Child. 47:261 (Feb.) 1934.

[24] Wolff, J. A.: Premature and newborn infants. A seminar report, Pediatrics 20:143 (July) 1957.

[25] Blackfan, K. D., Diamond, L. K., Leister, C. M.: Atlas of the blood in children. The Commonwealth Fund. New York 1944. Schulman.[26]

[26] Schulman, I., Smith, C. H., Stern, G. S.: Studies on the anemia of prematurity. A.M.A. J. Dis. Child. 88:567 (Nov.) 1954.

with increasing extrauterine age at least during the first three days of life.[27]

The full-term newborn has a greater percentage of adult hemoglobin than does the premature. This is not surprising and indicates that the replacement of the embryonic pigment begins in advance of birth. This replacement probably occurs very early, for Schulman found that even in a premature of less than 1,000 grams, there was at least ten per cent of adult hemoglobin present. The production of fetal hemoglobin probably continues to occur after birth because it can be found after 120 days. Although the premature infant has a higher percentage of fetal hemoglobin at birth it tends to lose it at an earlier age than the full-term infant. Methemoglobin is also higher in the premature infant. Kronitz found that it constituted 2.3 per cent of the total hemoglobin during the first week of life as compared to 0.47 per cent in the mature newborn.[28]

The blood volumes of 38 prematures were studied by Schulman and his group.[26] They found that at birth the blood volume was high (about 108 ml/kg) then steadily fell so that at about the seventh week it averaged 73 ml per kg body weight. The volume of the red cells was estimated to be about 46 ml per kg body weight at birth and 16 ml per kg at the seventh week.

Hemorrhagic manifestations are quite frequent in the newborn period. The bleeding may be from the mouth, rectum, or cord or appear as ecchymotic areas in the skin. Many times this is only transitory and does not endanger the well-being of the newborn infant; in a small number of instances it may indicate a serious condition. Formerly, this picture was called hemorrhagic disease of the newborn and was said to occur more frequently in prematures. We now consider many cases reported in the older literature to be due to other causes such as erythroblastosis, sepsis, anoxia, or trauma instead of to coagulation disturbances. In considering this bleeding tendency in the newborn, two factors have to be considered: inability of the blood to coagulate properly, and the integrity of the vascular wall.

It is generally accepted that the platelets are not at fault in the delayed blood clotting of this disease. The bleeding time during the first five days of life is increased as compared to the first day (4 min vs. 2 min). The clotting time is variable and correlates with the prothrombin level.

Actually, there is a tendency for all the elements concerned in the coagulation of the newborn blood to diminish slightly during the first few days of life. The prothrombin at birth is 70 to 80 per cent, similar to the mother's value at the time of delivery. This decreases to 20 per cent by the third day, then returns to adult values. The fibrinogen is low for the first five days of life, then rises. Calcium falls from birth to the fifth day, then increases.

The low prothrombin values are attributed to a deficiency of vitamin K. The presence of low prothrombin, however, does not necessarily cause hemorrhage. Undoubtedly there are other blood factors involved in the problem. It is believed that there is a decreased plasma thromboplastin component, plasma thromboplastin accelerator, and proconvertin. All are related to the low prothombin content and are returned to normal in the full term infant by vitamin K. Prematures with the same defects show a lesser response to vitamin K.[29]

There is probably some factor which causes a defect in the walls of the blood vessels which is involved in the hemorrhagic disease of the newborn but as yet no information seems to have been published to date.

Renal Function

The constancy of the internal environment is largely the function of the kidney. During fetal life the placenta does most of this regulation but the burden falls on the kidney within a few minutes of birth. Many years ago, Eckhardt considered that no new glomeruli were formed after birth. Potter and Thiersten found that developing glomeruli could no longer be found after an infant had reached a weight of 2,100 to 2,500 grams.[30] This means that prematures have a functional handicap which full-term infants do not. Also, in the neonate the cortex makes up a much smaller proportion of the kidney than it does in later life. In the adult the glomerular tuft is covered with thin epithelium, but in fetal life the tuft is covered with full columnar cells. At full term, the cells are less conspicuous, and cuboidal, and may not cover the tuft.

In general, then, it can be stated that the newborn kidney is a less effective organ than that of the older child or adult and that the premature kidney is less effective than the full-term infant's. The work in studying renal function involves certain clearance tests; however, electrolyte clearances do not have the same significance as do clearances measuring discrete kidney functions. This is easily realized when it is remembered that many nonrenal factors such as intake, retention, hormones, and metabolic demands all affect the rate of renal excretion of electrolytes.

[27] Fowler, R. S.: Extramedullary erythropoiesis in the livers of premature infants, A.M.A. J. Dis. Child. 91:245 (Mar.) 1956.

[28] Kravitz, H., Elegant, L. D., Kaiser, E., Kagan, B. M.: Methemoglobin values in premature and mature infants, A.M.A. J. Dis. Child. 91:1 (Jan.) 1956.

[29] Abelli, A. J., Banus, V. L., de Lamerens, S., Rozengroig, S.: Coagulation studies in the newborn period, A.M.A. J. Dis. Child. 94:589 (Dec.) 1957.

[30] Potter, E. L., Thierstein, S. T.: Glomerular development in kidney as index of fetal maturity, J. Pediatrics 22:695 (June) 1943.

Although not definitely established, it is thought that renal function in young infants correlates best with surface area. It has been proposed that glomerular function in young infants is more fully developed than tubular function and that gradual improvement in function after the newborn period is due to improvement in the tubules. The late development of the loop of Henle is generally considered to be responsible for this. In consequence the infant kidney is unable to vary tubular reabsorption of water and salt. There does not appear to be any inability to secrete posterior pituitary antidiuretic hormone or adrenocortical hormones, though it is possible that the infant lacks the power to adjust the amounts secreted with any precision.

The over-all functional capacity of the newborn kidney is one of little flexibility. It excretes water, sodium chloride, and urea, but if their relations are altered in the plasma, the kidney seems unable to make any great effort to restore the balance. The clinical implications of this inability are great, such as in infant feedings and in the treatment of dehydration and acidosis.

In prescribing formulae, the minimum and maximum quantity of water which should be provided for excretion of urinary solutes will be determined by the ability to concentrate or dilute the urine. Furthermore, the quantity of urine solutes requiring excretion will depend upon the electrolyte and protein content of the diet and on the proportion of solutes retained for growth or replacement. They will also vary with extrarenal losses via sweating, vomiting, and diarrhea.

Prematures getting cow's milk with added carbohydrate and weighing 2.0 kg excrete about 15 to 20 milliosmols of total urinary solute per kg per day. In older infants and adults this quantity of solute could be excreted in 10 to 15 ml of water per kg because they can concentrate up to 1,400 milliosmols per liter. This 10 to 15 ml per kg would represent the minimum renal water requirement under these conditions. Under the same conditions the premature infant can concentrate these urines to only about 700 milliosmols/liter, so that twice the volume, or 20 to 30 ml of water per kg, are needed.[31]

Since both prematures and adults can dilute urine to about 50 milliosmols liter it follows that the maximum quantity of water which could be excreted with 15 to 20 milliosmols of urine solute is about 300 to 400 ml/kg/day.

The question of water and electrolyte balance in the premature infant is clouded by controversy as to what the normal values are. It has been recognized that premature infants have a metabolic acidosis. This was perhaps first pointed out by Ylppö.[32] This is attributed to the increase in inorganic and organic acids. Darrow related the production of acidosis in premature infants to immaturity of the kidney and to the electrolytes in the formula.[33] Reardon *et al.*, from their studies previously mentioned, did not feel that high chloride intake from the ingested milk was the cause of the acidosis.[17] A theory acceptable to many is that there is a partial anaerobic catabolism of carbohydrate leading to decreased production of carbon dioxide and increased formation of organic acids, resulting in an acidosis.

To summarize, the newborn infant has relatively more water in the body than the adult, with the greater part of it extracellular.[34] The predominant extracellular electrolyte is potassium. In comparison, the serum has less protein and bicarbonate but more chloride and phosphates. The sodium and osmotic pressure of the serum are similar to adult values. The osmotic pressure of the urine is 500 mOsm per liter by the second day then falls to 100 mOsm per liter on the fourth day and remains so to the tenth day. Average daily urine output is about 50 ml with gradual increase to 200 ml by the tenth day. Functional maturity of the glomeruli may take ten weeks to six months with the tubules taking a still longer period. Because clearances are low, consideration must be given the load placed on the immature kidney.

The Adrenals

The adrenal gland of the newborn is very unlike that seen in later life. The newborn adrenal glands comprise approximately 0.29 per cent of the birth weight compared with approximately 0.14 per cent of the birth weight in adults. At the time of birth the infant adrenal cortex has two histologically distinct zones; an outer adult-type cortex consisting of a zona glomerulosa and short straight strands of zona fasciculata, and a much larger inner zone. The inner region comprises 80 per cent of the entire newborn adrenal gland. This region is variously called fetal transitional zone, internal cortex, fetal androgenic zone or fetal cortex. It consists of large cells and may be the equivalent of the zona reticularis. Although the adrenal is primarily a homeostatic system in the adult, its function in the fetus is unknown and may be entirely different.

During fetal life, the fetal zone grows very rapidly. At the termination of pregnancy it involutes, and in two or three months is largely destroyed, and, as far as can be ascertained, does

[31] Pratt, E. L., Bienvenu, B., Whyte, M. M.: Concentration of urine solutes by young infants, Pediatrics 1:181 (Feb.) 1948.

[32] Ylppö, A.: Beitrag zur Azidosis bei Neugeborin, Acta Paediat. 3:235, 1924.

[33] Darrow, D. C., da Silva, M. M., Stevenson, S. S.: Production of acidosis in premature infants by protein milk, J. Pediat. 27:43 (July) 1945.

[34] McCance, R. A.: Renal function in early life, Physiological Reviews 28:331 (July) 1948.

not reappear in later life.[36] With involution of the fetal zone the adult-type cortex grows rapidly. Involution does not seem to be related to any particular stage of physiologic development, for it occurs just as rapidly in the premature as in the full-term infant. There is thought to be some relationship between the fetal cortex, the fetal pituitary and the placenta. In anencephalics or in other fetuses with some severe anomaly of the nervous system the adrenals are found to be small. However, if an anencephalic monster is aborted early in gestation before the fourth month, the fetal zone is said to be present and normal. It is therefore postulated that the placenta plays a role in maintaining the fetal zone. Experimentally, human chorionic gonadotropin and ACTH have been used without success to see if the fetal zone could be preserved. Lanham has been unable to demonstrate 17-ketosteroids in the human fetal adrenal gland.[35] Gardner measured 17-ketosteroids in the blood of newborns and found that during the first two days of life, the levels were equivalent to those of adults but rapidly fell during the first week.[36] Prematures maintained elevated levels in their plasma for several weeks. Cranny studied the urinary excretion of Porter-Silber chromogens by premature infants.[37] They are excreted in the urine as free and conjugated forms and are thought to be a measure of 17-hydroxycorticosteroids. It was found that excretion varied with increasing age and also with increasing urine volume output. It is not known how much this might be a reflection of renal immaturity. On the other hand, there is a fall in excretion of urinary 17-ketosteroids during the first several weeks. Possibly 17-ketosteroids are synthesized by the fetal cortex, and corticosteroids are produced by the adult-type cortex.

It is not thought that prematures have impaired adrenal activity. Levine has given large doses of ACTH, as much as 100 mg per day, and found that the infants develop an enormous appetite and also tend to cry persistently.[38] He found that they would take up to 200 or 300 calories per kg and be unsatisfied. They would also lose weight despite the large caloric intake. It is not recommended that ACTH be so used, because death can also occur. Silverman found that ACTH in excess of 5 mg per kg per day inhibited growth in prematures.[39] In several who died he found that osteogenesis and chondrogenesis were retarded. The livers were found to be filled with glycogen and the adrenals were markedly enlarged.

[35] Lanham, J. T.: Fetal-maternal endocrinology in late pregnancy. Physiology of prematurity. Josiah Macy, Jr. Foundation, 1956.
[36] Gardner, L. I.: Adrenal function in the premature and newborn. Pediatrics 17:414 (Mar.) 1956.
[37] Cranny, R. L., Cranny, C. L.: Urinary excretion of Porter-Silber chromogens by premature infants. A.M.A. J. Dis. Child. 95:401 (Apr.) 1958.
[38] Levine, S. Z.: Physiology of prematurity. Josiah Macy, Jr. Foundation, 1956.
[39] Silverman, W. A.: Physiology of prematurity. Josiah Macy, Jr. Foundation, 1956.

Nutrition

The physiology of the newborn gastrointestinal tract is well known and will not be elaborated on. It is generally accepted that newborns and even prematures are able to assimilate proteins well. Positive nitrogen balance usually occurs by the end of the first week. The plasma proteins are about 6 gm per cent with the deficiency mainly in globulin fraction. Prematures have a more marked hypoproteinemia. The minimal requirement of protein is that which will permit normal weight gain, and varies with age in proportion to rate of growth. The estimated requirement for a full-term infant is approximately 2.2 gm per kg per day, possibly 3 gm per kg per day for prematures. Most people prefer to give 25 per cent more than the calculated minimum requirement. This requirement is dependent upon its content of essential amino acids; however, utilization depends not only on total quantity available but in accordance with certain ratios, so that specific supplementation may lead to amino acid imbalances. Because time is required to enable the body's metabolism to adapt to a high protein load, the protein content of any diet should be increased slowly. Also, there may be a maximal protein intake which an infant can tolerate.

The average premature infant absorbs fat poorly. He can absorb only 60 to 70 per cent of ingested butterfat compared with the 90 per cent absorbed by the full-term infant. Olive oil is said to be absorbed to about 70 to 90 per cent. It is helpful to promote fat absorption in order to provide essential fatty acids and fat-soluble vitamins. Balance studies show that absorption can be improved by selecting short-chain fats or fats with many unsaturated carbon bonds. Holt believes in giving fats in the form of corn oil to provide a source of calories. He believes that even if only 50 per cent of the ingested fat is absorbed, gram for gram more calories can be derived from fat than from carbohydrates. Levine feels that it is not economical to use fat which is poorly utilized when carbohydrate and protein are known to be more efficiently utilized. The true requirement for fat is thought to be negligible.

The actual requirement for carbohydrate is not known but it has been shown to be well absorbed and well utilized. Glucose and sucrose tagged with C_{14} and C_{13} and given to premature infants showed 99 per cent absorption and within two hours 90 per cent was expired as carbon dioxide.[40]

Studies by Gordon *et al.* comparing premature infants fed on a half-skimmed cow's milk mixture with those fed on isocaloric amounts of human milk, showed that the former gained more weight per unit body weight per day during the first

[40] Holt, L. E., Jr.: Premature and newborn infants. Report of a seminar. Pediatrics 20:143 (July) 1957.

month of life than the latter infants.[41] It was suggested that the greater weight gain was due to higher levels of protein or ash, or both. Kagan and co-workers studied 156 prematures with birth weights from 1,000 to 2,000 grams.[42] They found that at both high and low levels of caloric intake a good correlation existed between increment in weight gain and increment in ash intake. They felt that increasing caloric intake by increasing the volume of unmodified cow's milk or by increasing the proportion of unmodified cow's milk protein led to a relatively large intake in ash. This is probably due to the low rate of renal solute excretion of the premature infant, as noted by Barnett, which would lead to electrolyte and water retention on a high mineral intake.[43] Pratt and Snyderman showed that when two groups of prematures were fed isocaloric amounts of an evaporated milk formula with and without added carbohydrate, the formula without added carbohydrate had an 85 per cent increase in renal solute load and a 1.42 times the amount of ash.[44] It has been noted that, when the diet is changed from human milk to cow's milk, there is a lag in renal excretion of sodium and chloride ingested which accompanies the large weight gain. Rubin found that when premature infants were fed milks of high protein concentration, the weight gain was greater when the intake of carbohydrate was increased regardless of whether the comparative milks were isocaloric or isonitrogenous.[45]

Wallace, Weil, and Taylor studied the composition of growth of the albino rat on high protein–high electrolyte, on high protein–low electrolyte, on low protein–low electrolyte, and low protein–high electrolyte diets.[46] When the animals doubled their weaning weights chemical analyses were performed on the whole bodies. The animals on the low protein intakes grew significantly less and their bodies contained more fat. The principal difference resulting from variation in intake was in the quantity of the skeletal constituents in the various groups. Those on low protein rations contained more calcium and phosphorus on a unit basis than did the high-protein fed animals. On the high protein intakes accretion of skeletal minimals was dependent upon the level of electrolyte intake, being higher in the high-electrolyte-fed animals. In the low-protein-fed animals this accretion was less affected by level of electrolyte intake. Only in the animals on the high electrolyte diets did increased protein intake result in increased protein content of the body.

Premature infants require as a minimum volume of fluid about 125 ml per kg per day. Volumes below this lead to a negative fluid balance. Optimal fluid intake, about 140 ml per kg per day, is given when the infant is stabilized. Daily caloric requirement is about 120 calories per kg per day. Newborn premature infants are often starved initially for fear of aspiration and to aid in losing tissue edema. There is thus a tendency for the infant to develop a metabolic acidosis from breaking down of body stores for energy, with consequent accumulation of organic acids. Also, since water is required for renal solutes to be excreted, it seems reasonable that prolonged starvation may be harmful. Most centers initiate feeding with the loss of clinical edema.

Summary

Certain aspects of the physiology of the premature infant have been discussed with the aim of using a rational approach in the care of the infant based on knowledge of the normal processes. The initiation of the first breath and the mechanisms of breathing are discussed with emphasis on respiratory dysfunction. The anemia of prematurity is reviewed in terms of etiology and possible modes of therapy. The renal and adrenal functions of the premature and newborn infants are briefly touched on. Certain concepts of infant feeding are given based on metabolic studies and the possibility of electrolyte imbalances resulting from an overloading of an immature kidney.

Summario in Interlingua

Le infante prematur require frequentemente plus adjuta que lo que le medico pote offerer. Le problema es immaturitate physiologic. Le pulmones de infantes prematur non ha alveolos in numeros sufficiente. Illos suffre de un manco de histos elastic. Le prematuro tende a respirar irregularmente. Morbo de membrana hyalin pote occurrer. Le sanguine in le circulation es immatur, e anemia es un occurrentia commun. Manifestationes hemorrhagic pote occurrer. Le ren del prematuros es ancora minus efficace que le ren de neonatos normal. Le glandula adrenal del infante prematur ha ancora un multo grande "cortice fetal" e un relativemente tenue "cortice adulte," ben que le signification de iste facto non es cognoscite. Le absorption gastrointestinal de grassia es etiam inefficace in infantes prematur.

[41] Gordon, H. H., Levine, S. Z., McNamara, H.: Feeding of premature infants, a comparison of human and cow's milk, A.M.A. J. Dis. Child. 73:422 (Apr.) 1947.

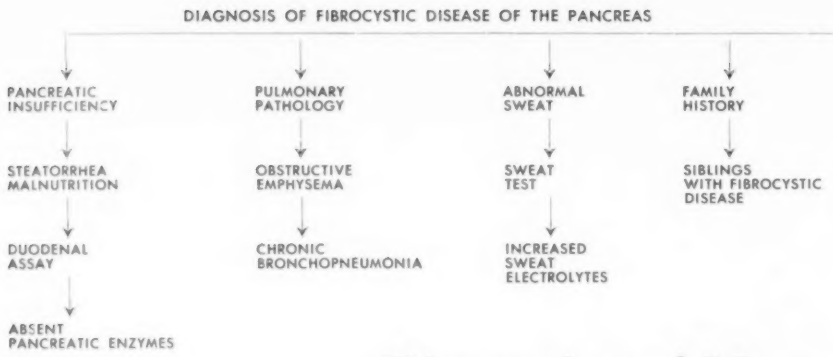
[42] Kagan, B. M., Hess, J. H., Lundeen, C., Shafer, K., Parker, J. B., Stigel, C.: Feeding premature infants. A comparison of various milks, Pediatrics 15:373 (Apr.) 1955.

[43] Barnett, H. L., Visterdal, J.: The physiologic and clinical significance of immaturity of kidney function in young infants, J. Pediat. 42:99 (Jan.) 1953.

[44] Pratt, E. L., Snyderman, S. E.: Renal water requirements of infants fed evaporated milk with and without added carbohydrate, Pediatrics 11:65 (Jan.) 1953.

[45] Calcagno, P. L., Rubin, M. I.: Effect of added carbohydrate on growth, nitrogen retention and renal water excretion in premature infants, Pediatrics 13:193 (Mar.) 1954.

[46] Wallace, W. M., Weil, W. B., Taylor, A.: The effect of variable protein and mineral intake upon the body composition of the growing animal. Water and electrolyte metabolism in relation to age and sex. Ciba Foundation Colloquia on Aging. Volume 4, 1958.



Diagnosis and Management of Fibrocystic Disease of the Pancreas

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CYSTIC FIBROSIS of the pancreas may be defined as a congenital familial disease characterized by dysfunctions of many of the exocrine glands. The most obvious effects are pancreatic deficiency with characteristic morphologic changes in that organ, susceptibility to chronic bronchitis, and a high concentration of electrolytes in sweat. Other exocrine glands may give functional or morphologic evidence of abnormality. The basic defect is unknown. Death occurs in infancy or childhood in a number of cases.

A variety of names have been given to the disease: congenital pancreatic disease, congenital steatorrhea, congenital pancreatic steatorrhea, familial pancreatic fibrosis, cystic fibrosis of the pancreas, fibrocystic disease of the pancreas, mucoviscidosis, dysporia enterobronchopancreatica congenita familiaris, etc. The most widely used terms are cystic fibrosis of the pancreas and fibrocystic disease of the pancreas which, although they fail to describe the disease completely, are generally accepted and relatively simple.

Diagnosis

Making a diagnosis of fibrocystic disease of the pancreas involves the utilization of the family history, clinical symptoms and laboratory findings.

Family History

The presence of siblings with the same condition, or presence of abnormal sweat electrolytes in siblings or parents without clinical symptoms of fibrocystic disease of the pancreas, are of diagnostic assistance; however, their absence does not rule out the disease.

Clinical Manifestations

The infant appears normal at birth, although the birth weight is, on the average, somewhat low. Prematurity occurs with normal frequency.

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The earliest recognizable clinical disturbance produced by pancreatic fibrosis is meconium ileus in the newborn infant. Ten to 15 per cent of infants born with cystic fibrosis of the pancreas have congenital intestinal obstruction due to meconium ileus. Tenacious, mucilaginous meconium plugs result from failure of liquefaction of meconium in the fetal intestinal tract because of grossly deficient or absent pancreatic enzyme.

Infants who recover from meconium ileus exhibit the later and more common manifestations of their persistent pancreatic deficiency which results in poor intestinal digestion and absorption. Almost always before the sixth month, and often before the fourth week, the abnormality of the stools becomes evident. A small proportion of cases develop severe diarrhea, which may respond to a low fat diet with vitamin supplements. The majority have three to five large fatty, bulky, foul

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smelling stools. After infancy stools are not excessive in number and may have been considered normal.

Failure to gain weight in spite of an adequate dietary intake is usually apparent early.

Respiratory infection is an almost constant feature of the disease. In most cases cough begins in the second to fourth month, often initiated by coryza. The cough may be present from birth and is apparent before the age of six months in most cases. It may first be merely an occasional dry hack or may occur in paroxysms resembling those in pertussis, and may be accompanied by cyanosis and vomiting. Difficulty in relieving the tracheobronchial tree of its contents is obvious; respirations are audible, and the obstruction is sometimes so persistent as to produce a laryngeal stridor. Obstructive emphysema and tachypnea soon appear and may be the first recognized symptoms.

The infection may progress to a bronchopneumonia in the course of a few days or weeks or may remain static until a subsequent coryza leads to a more severe bronchitis. During early childhood the less fortunate of these patients present a distressing picture of obstructive emphysema, respiratory distress, cyanosis, and paroxymal cough. Suppurative bronchitis is still the major cause of death because the emergence of a resistant organism often renders antibiotic therapy ineffective. Death may result either from asphyxia because the trachea becomes filled with mucopurulent material, or from right heart failure.

The earliest radiologic signs of pulmonary disease are emphysema with narrowing of the cardiac shadow, depression of the diaphragm, and increased bronchovascular markings, also small or large areas of atelectasis especially in the right upper and middle lobes.[1] Bronchograms following the instillation of lipiodol are of doubtful value in diagnosis, since bronchiectasis of the larger bronchi is unusual. Mild fusiform dilatation of the larger bronchi may occur in long-standing severe disease. Occasionally the saccular dilatation of the terminal bronchioles may be well demonstrated.

Most patients are small for their age and retardation is obvious. In those children who survive to later childhood or adolescence, certain complications of the disease become more prominent. Among those are multilobular cirrhosis of the liver, peptic ulcer, hematemesis, and spontaneous mediastinal emphysema and pneumothorax.

Laboratory Studies

Laboratory studies may show abnormal findings in stool, duodenal juice, blood or plasma and sweat electrolytes.

Stool trypsin. Measurement of stool trypsin is a simple test for the proteolytic activity of stool, done by determining its ability to liquefy gelatin. This test is negative in patients with fibrocystic disease of the pancreas because of the absence of stool trypsin,[2] but because of the possibility of a false positive test due to liquefying bacteria, this test is not reliable.[3]

Fecal fat. Microscopic examination for excess fat in stool is fairly reliable as a screen test in those patients who have pancreatic deficiency and are given a normal diet. This method has no value when the dietary fat is restricted.

Duodenal juice. Assay of pancreatic enzymes in the duodenal juice is a widely used diagnostic procedure in patients with pancreatic fibrosis. Amylase, trypsin, and lipase are all lacking or present in minimal amounts, depending on the degree of pancreatic deficiency. The determination of trypsin was found to be the most reliable means of diagnosis.

Activity of trypsin in the duodenal juice is measured in approximate terms by the digestion or liquefaction of gelatin by various dilutions of duodenal juice. The duodenal juice in healthy children contains 400 to 40 tryptic activity units per cc. Patients with marked pancreatic deficiency will have less than 20 tryptic activity units.[4]

The other method is determination of the viscosity of the duodenal juice, which is abnormally high in patients with fibrocystic disease of the pancreas. These patients also exhibit a characteristic difference in water solubility of the mucoprotein fractions of duodenal fluid. The mucoproteins in the duodenal juice of fibrocystic patients are insoluble in distilled water, whereas the mucoproteins of normal individuals are easily soluble. This abnormal mucoprotein fraction cannot be made soluble by adding trypsin, but can be made soluble by heating and adding sodium hydroxide.[4] It has also been found that there is an abnormal electrophoretic pattern of duodenal juice in patients with fibrocystic disease of the pancreas.

Blood or plasma. Fasting blood levels of total lipids are within the normal range or somewhat low. The iodine number of the fatty acids present in plasma and the total serum cholesterol are abnormally low. The fasting serum vitamin A, serum carotene, and plasma tocopherol are low.

Poor absorption of vitamin A is evidence of failure of fat digestion due to absence of pancreatic lipase. Absorption curves with oily vitamin

[1] Cooke, R. E.: Cystic fibrosis of the pancreas, Southern Med. J. 51:208 (Feb.) 1958.

[2] Shwachman, H., Patterson, P. R., and Laguna, J.: Studies in pancreatic fibrosis: Simple gelatin film test for stool trypsin, Ped. 4:222 (Aug.) 1949.

[3] Johnson, D. E.: Studies on gelatin liquefying properties of gelatin bacteria with particular reference to laboratory diagnosis of cystic fibrosis of the pancreas, Ped. 6:35 (Sept.) 1950.

[4] Harrison, G. M., and Taylor, F. M.: Diagnosis and management of fibrocystic disease in infants and children, Texas St. J. of Med. 54:296 (May) 1958.

A preparation are flat but with the alcoholic form of vitamin A they are normal, in contrast to the results in idiopathic celiac disease in which both forms of vitamin A are poorly absorbed.

Sweat electrolytes. The normal sweat chloride level is from 4 to 80 mEq with an average of 32 mEq. The range for the patient with fibrocystic disease of the pancreas is from 60 to 160 mEq with a mean of 106 mEq.

The normal sweat sodium is from 10 to 120 mEq with an average mean of 59 mEq. The range for patients with cystic fibrosis of the pancreas is from 80 to 190 mEq with a mean of 133 mEq.

The difference of the mean value for potassium content of sweat in the normal child and in the patient with cystic fibrosis is less but still is significant.

The mean volume of sweat is not significantly different in the normal child and the patient with cystic fibrosis.[5]

A study of sweat electrolytes in all patients with fibrocystic disease of the pancreas has shown that there are:

A. Abnormally high sweat chloride and sodium in all patients with anatomical changes of fibrocystic disease of the pancreas at autopsy.[5]
B. High sweat chloride and sodium in 90 per cent of living patients with the pulmonary picture characteristic of fibrocystic disease of the pancreas and also low or absent tryptic activity in the duodenal juice.[6]
C. High sweat electrolytes in some of the close relatives (parents or siblings) of known patients whose relatives have the typical pulmonary picture but entirely normal pancreatic function.
D. High sweat chloride and sodium in some of the close relatives of known patients, whose relatives have neither generalized obstructive emphysema with secondary chronic bronchopneumonia nor low duodenal tryptic activity.[6]
E. Pancreatic deficiency, chronic pulmonary disease and the administration of certain drugs do not per se affect the electrolyte composition of the sweat. Also administration of pancreatin or any kind of antibiotics do not affect the sweat electrolyte concentration of the patients with cystic fibrosis of the pancreas.[7]

In the great majority of patients all criteria are present. However, if the pancreatic function is normal, the combination of abnormal sweat and typical pulmonary manifestations is, to put it conservatively, very suggestive of the disease. Conversely, the combination of pancreatic deficiency and high normal or abnormal sweat electrolytes without any lung changes is found at times in some patients. Patients who have pancreatic deficiency and the pulmonary changes but low normal sweat electrolytes have not been observed. The combination of low normal sweat electrolytes and normal pancreatic function is also theoretically possible, but highly unlikely and in this latter case diagnosis would be virtually impossible.

The combination of cirrhosis of the liver with portal hypertension and chronic pulmonary disease is very suggestive of cystic fibrosis even in the absence of pancreatic involvement. Here again an abnormal or high normal sweat test will support the diagnosis.

The differential diagnosis varies somewhat with the age.[8] In early infancy the poor gain in weight may be wrongly attributed to malnutrition. The cough may simulate pertussis, and the difficult breathing has been considered laryngeal stridor. In older infants the usual erroneous diagnosis is celiac disease. The differentiation between the steatorrhea of celiac disease and that of cystic fibrosis can be made an office procedure with the aid of the microscopic test for fecal fat on a normal fat intake. After a month's trial of a therapeutic celiac diet most patients of both groups show clinical improvement. However, on return to a normal diet the children with celiac disease show rapid clinical deterioration but slow return of steatorrhea, while those with cystic fibrosis tolerate the normal diet well but have immediate return of steatorrhea.

Prognosis

The prognosis has greatly improved in the past 20 years. Prior to the general clinical recognition of the disease the majority of patients died during their first year. More recently the majority of deaths have occurred between the ages of three and five years. However, in the Babies' Hospital more than half of the total series of over 400 patients are living at various ages up to 21 years. There have been few deaths during adolescence, and most older patients show slow or no progression of pulmonary changes. Most of them have some clubbing of the fingers and many have moderate reduction of vital capacity. The future of the individual patient is unpredictable, especially in infancy. The diagnosis carries with it a serious but far from hopeless prognosis.[8]

Therapy

The pancreatic deficiency is irreversible and the susceptibility to respiratory disease and to heat prostration is also permanent. Much can be done

[5] di Sant'Agnese, P. A.: Fibrocystic disease of the pancreas, Ped. 15:683 (June) 1955.
[6] Mauer, A. M., and West, C. D.: A simple method for collection and analysis of sweat for chloride, Am. Med. Assoc. Dis. of Child., 29:160 (Aug.) 1956.
[7] di Sant'Agnese, P. A., Darling, R. C., Perera, G. A., Shea, Ethel: Abnormal electrolyte composition of sweat in cystic fibrosis of the pancreas, Ped. 12:549 (Nov.) 1953.
[8] Anderson, D. H.: Cystic fibrosis of the pancreas, J. of Ch. Dis. 7:58 (Jan.) 1958 and Therapy and prognosis of fibrocystic disease of the pancreas, Ped. 3:406 (1949).

in the prophylaxis and treatment of the sequelae of these basic abnormalities.

Respiratory infection dominates the picture in most cases and is the usual cause of death. Once well established, the pulmonary lesions tend to persist and increase. Prophylactic dietary measures may delay the onset and development of pulmonary disease.

The use of a wide spectrum antibiotic is advisable. Patients with chronic pulmonary disease should be given one-fourth to one-half the usual therapeutic dosage, changing every four to six weeks with a different antibiotic, thereby minimizing the emergence of resistant strains of staphylococci and of gram negative bacteria (Pseudomonas aeruginosa, E. coli, and so forth). In superimposed infections patients are routinely given either oxytetracycline, chlortetracycline, or chloramphenicol daily, reserving erythromycin, novobiocin, and bacitracin for acute and more severe attacks of pneumonia and bronchitis. Cultures are carried out every two months in order to note any significant change in bacterial flora of throat or sputum, or alteration in sensitivity to antibiotic agents.[4]

Inhalation of an antibiotic aerosol may be of some help for the child who is more than four years old. Most of the antibiotics have been used by aerosol and inhalation is sometimes more effective than oral therapy.

Physical aids for the removal of material from the bronchial tree, such as postural drainage and negative pressure, as in the "cough machine," have been used. The results are more effective in older children, according to various reports.[1]

The essential objective of dietary therapy is to provide an abundance of calories and essential nutrients in spite of the digestive handicap. Hunger should be satisfied, and this may require as much as 200 calories per kilogram of body weight per day for small infants or 125 to 150 calories per kilogram of body weight per day for older children. The protein and carbohydrate should be high and an abundance of fat-soluble essential substances should be given.[9]

[9] Anderson, D. H.: Cystic fibrosis of the pancreas, J. of Ch. Dis. 7:58 (Jan.) 1958.

Opinions differ as to restricting nonessential fats. Moderate restriction is generally advised. Supplements of both fat-soluble and water-soluble vitamins should be given.

Synthetic pancreatic enzymes are given if there is major deficiency in pancreatic function; this has been known to improve the utilization of dietary fat and protein.

Pancreatin administration does not alter the course of a patient with severe chronic pulmonary disease. There is some evidence that it contributes to delaying the onset and progression of respiratory disease, probably by improving the general nutrition and resistance to infection.

Summary

Cystic fibrosis of the pancreas may be defined as a congenital, familial disease characterized by dysfunction of many of the exocrine glands. The most obvious effects are pancreatic deficiency, with characteristic morphologic changes in that organ; susceptibility to chronic pulmonary disease; and a high concentration of electrolytes in the sweat.

Diagnosis depends on suspicion, on certain presumptive clinical signs and symptoms, on family history, and on the demonstration of abnormal findings in secretions from the pancreas and cutaneous glands.

Proper treatment consists of using prophylactic antibiotics over long periods, nutritional aids and pancreatic enzyme.

Summario in Interlingua

Morbo fibrocystic del pancreas es le effecto de un congenite e probabilemente hereditabile dysfunction de un o plures del glandulas exocrin, notabilemente le eccrin glandulas sudoripare e le pancreas. Ileus per meconio in le neonato es un precoce signo characteristic. Chronic morbo pulmonar, typicamente initiate in le presentia de tusse durante le secunde e le quarte mense, es etiam commun e characteristic. Alte nivellos de natrium e de chloruro se trova quasi invariabilemente. Le prolongate administration de antibioticos es necessari pro prevenir infectiones pulmonar de character potentialmente mortal. Enzymas pancreatic pote esser utile.

Tests only become meaningful when the concept of the liver as a dynamic regulator of specific excretory functions and integrated metabolic activities is kept in mind.[1]

Liver Function in Infants and Children

With Special Emphasis on Liver Function Tests

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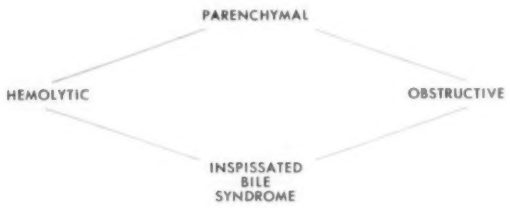
IT IS ASSUMED that liver function tests have less reliability and acceptance in pediatrics than in adult medicine. The immaturity of the neonatal liver, the dissociation and multiplicity of its functions, and large reserve capacity have made the interpretation of results difficult. Isolated studies into the metabolic processes of the liver have increased our knowledge of the normal expectations of this organ. Several excellent reviews have been written by Najjar[2] and Driscoll and Hsia.[3]

Liver function tests should be simple, sensitive, and specific. They are used (1) to determine the existence of hepatic disease of different types, (2) to determine the type and degree of involvement, and (3) to follow the course of the patient and to determine the progress (see Table 1). In pediatrics the problem of hepatic disease is particularly important in the newborn period, and seemingly, in this age period, liver function tests have the least value.

TABLE 1.—*Classification of Neonatal Jaundice.*

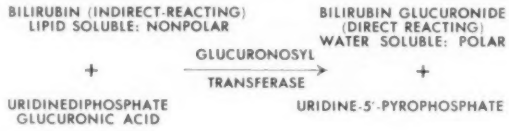


One of our chief aims is to measure the degree rather than the presence of hepatic dysfunction. The prevention of progressive hepatic damage which may result from obstructive jaundice and the importance of early surgical intervention for correctable abnormalities and, vice versa, the avoidance of unnecessary surgery in medical forms of neonatal jaundice, including inspissated bile syndrome (Patterson), give us our goal for an accurate diagnostic conclusion. The controversial topic of liver biopsy, open exploration, and cholangiography will be briefly discussed.

The Meaning of Physiologic Jaundice

The percentage of full-term and premature infants expected to develop physiologic icterus is surprisingly high.[4] The maximum serum bilirubin (7 mg per 100 ml) is attained in the full-term infant at two days of life. In the premature newborn this is attained at the end of four days of life (11 mg per 100 ml).[5] This is perhaps the first indication of hepatic immaturity of the newborn infant.[6] The more recent theories of bilirubin metabolism suggest the deficiency of certain enzymes which aid in the conversion of indirect to direct forms of bilirubin.

TABLE 2.



Bilirubin (indirect-reacting) in the presence of the enzyme, glucuronosyl transferase, is conjugated with glucuronic acid to form bilirubin glucuronide (direct-reacting) (see Table 2). The bilirubin glucuronide is actually two pigments, pigment I and pigment II.[7] In liver disease, the conversion of pigment I to pigment II diminishes. Thus, in acute hepatitis, pigment I is said to be predominant.

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[1] Hanger, F. M.: The meaning of liver function tests, Am. J. Med. 16:565 (Apr.) 1954.

[2] Najjar, V. A.: The metabolism of carbohydrates, fats and bile pigments by the liver and the alterations in hepatic disease; a review of recent advances, Pediatrics 15:444 (Apr.) 1955.

[3] Driscoll, S. G., and Hsia, D. Y. Y.: The development of enzyme systems during early infancy, Pediatrics (Supp.) 22:801 (Oct.) 1958.

[4] Weech, A. A.: The genesis of physiologic hyperbilirubinemia, Adv. Pediat. 2:46 (1947).

[5] Gellis, S. S., and Hsia, D. Y. Y.: Liver function tests in infants and children, Ped. Clin. of N. Am. (Feb.) 1955.

[6] Holman, G. H.: Studies on physiologic hyperbilirubinemia of negro and white premature infants, Pediatrics 22:1115 (Dec.) 1958.

The solubility of indirect-reacting bilirubin in lipids may explain the absence of this form of bilirubin in urine and its affinity for brain tissue.

This glucuronosyl transfer system appears to operate in many other metabolic and detoxification processes of the liver, and has formed the basis for some tests described later. Driscoll and Hsia[3] in their recent review discussed the experimental studies supporting the idea of relative hepatic immaturity of the glucuronosyl transfer system. They also stated that the deficiency of this system may be responsible for the so-called hereditary diseases designated as "familial nonhemolytic jaundice" or several of the "inborn errors of metabolism."

Hyperbilirubinemia of the newborn infant is actually a "transient immaturity" of this enzyme system.

The Preferred Tests in Pediatrics

The composite of statements made heretofore affords a classification of liver function tests in accordance to the general functions of the liver.

A. The metabolism of bile pigments: excretion.
1. **Serum bilirubin: direct-reacting and total.**
2. **Urine bilirubin and urobilinogen.**
3. **Stool bilirubin and urobilinogen.**

B. The metabolism of serum proteins: synthesis.
1. **Flocculation tests: Thymol turbidity, thymol flocculation, cephalin-cholesterol flocculation, zinc sulfate flocculation, etc.**
2. **Albumin and globulins, A/G ratio, electrophoresis of proteins.**
3. **Prothrombin time.**

C. The metabolism of carbohydrates: glycogenesis, degradation, synthesis, storage, and external hormonal-enzymatic influences.
1. **Glucose tolerance test.**
2. **Galactose tolerance test.**

D. The metabolism of fats: excretion, synthesis, esterification, degradation.
1. **Cholesterol.**
2. **Cholesterol esters.**

E. Miscellaneous
1. **Enzymes: alkaline phosphatase, transaminases: excretion.**
2. **Bromsulphalein dye test, I^{131} rose bengal: excretion.**
3. **Needle biopsy and surgical exploration.**

Full details concerning their historical significance, and methods of analysis can be found in many reviews and laboratory manuals (Green,[8] Wolman,[9] Gellis and Hsia,[5] and Snell[10]). These remain outside the scope of this review.

The Metabolism of Bile Pigments

The presence of bile pigments in the stools of the newborn is not uncommon in the first few days of life.[11] However, they soon disappear with the introduction of the intestinal flora.

The absence of bile pigments in the stool gives the characteristic clay-colored stools, as seen commonly in the obstructive forms of jaundice. Generally, three conditions are present with stools of this form: congenital biliary atresia, neonatal hepatitis, and the inspissated bile syndrome.[12] According to Gellis and Hsia, the serum bilirubin is most useful in differentiating these three conditions. The level of the initial bilirubin and the periodic changes by serial determinations should be plotted against the course of the patient. In their evaluation of 156 patients with prolonged obstructive jaundice, the authors found the initial bilirubin level in inspissated bile syndrome to range from 10 to 30 mg per 100 ml. For either congenital biliary atresia or neonatal hepatitis, this level is 5 to 10 mg per 100 ml. The bilirubin levels show a steady upward trend in congenital biliary atresia, a rapid decline in inspissated bile syndrome due to erythoblastosis, and either a decline or variability of levels in neonatal hepatitis. Marked increases in the direct-reacting bilirubin suggest the presence of inspissated bile, whereas moderate elevations suggest the other two forms. According to Watson's ratio, the ratio of direct-reacting to indirect-reacting times 100, a value greater than 40 per cent may indicate the regurgitation form of jaundice as against parenchymal or hemolytic jaundice. The finding of urinary bilirubin is characteristic of all three forms of obstructive jaundice, and may reflect the degree of obstruction present.[13] Prior recommends examination of urine for urobilinogen at frequent intervals.[14]

DR. HO

The Metabolism of Proteins

Investigative works in this field have shown that levels of total proteins, albumin and globulins are constantly changing from fetal life to term, and thereafter. The concentrations of total proteins, albumin, and gamma-globulin increase steadily throughout fetal life, while the alpha and beta-

[7] Billing, B. H., and Lathe, G. H.: Bilirubin metabolism in jaundice. Am. J. Med. 24:111 (Jan.) 1958.

[8] Green, C. H.: Liver and biliary tract; a survey of tests for hepatic function. Arch. Int. Med. 86:743 (Nov.) 1950.

[9] Wolman, I. J.: Laboratory Applications in Clinical Pediatrics, The Blakiston Division. New York: McGraw-Hill Co., Inc., 1957, pp. 472-497.

[10] Snell, A. M.: Interpretation of liver function tests, Gastroenterology 34:675 (Apr.) 1958.

[11] Snelling, C. E.: Icterus neonatorum, J. Pediat. 2:399 (Apr.) 1933.

[12] Hsia, D. Y. Y., and Gellis, S. S.: Prolonged obstructive jaundice in infancy. III. Liver function tests. A.M.A.J. Dis. Child. 85:13 (Jan.) 1953.

[13] Ross, S. G., Waugh, T. R., and Malloy, H. T.: Icterus neonatorum, J. Pediat. 11:397 (Sept.) 1937.

[14] Prior, I.: Practical value of liver function tests and liver biopsy, N. Zealand M. J. 56:503 (Oct.) 1957.

globulins remain low.[15] The total serum proteins do not attain adult levels until approximately school age.

Conflicts arise from the variability and inconsistency of methods by which protein fractions are measured. As a group, they demonstrate gross changes in the albumin-globulin ratio.[16] Increases in gamma-globulin in liver disease have been attributed to stimulation of the Kupffer cells and the reticuloendothelial and mesenchymal systems, by the breakdown products of hepatic cells.[18]

In obstructive jaundice flocculation tests are frequently negative, especially in the newborn, and even in the presence of hepatic damage.[12] The flocculation tests become valuable aids in diagnosing diseases of parenchymatous involvement in later life, or according to Gellis and Hsia, after six months of age. The thymol flocculation test remains elevated during convalescence from hepatic disease. This test is related to the amounts of beta-globulin and its lipoprotein complexes. Therefore, it is generally elevated when the serum lipids are also elevated, as in chronic biliary cirrhosis. In infectious hepatitis the albumin values return to normal within one month at most, but the initial rise of gamma-globulin may persist for months.[18]

The cephalin-cholesterol flocculation test depends on the relative amounts of albumin and a labile complex of gamma-globulin. The former is inhibitory to the reaction between a specially prepared cephalin antigen and gamma-globulin. Increases in gamma-globulin and decreases in albumin or changes in its properties may result in positive cephalin-cholesterol reactions, as seen in early acute hepatitis. According to Salmon and Richman,[19] the cephalin-cholesterol is positive for several days after birth, probably due to the relative increase in gamma-globulin during this period. Positive reactions should also be expected in collagen diseases, acute glomerulonephritis, blood dyscrasia, iron deficiency anemia, tuberculosis, and many other diseases.[5]

These are some of the many reasons why some of these tests must be interpretated with the normal expectancy of the infant and child in mind.

Other tests in this group are quite similar in principle, with peculiarities of their own, and one should be acquainted with them if they are included in selected battery of tests.

The Metabolism of Carbohydrates

The intricate pathways of intermediary metabolism give us an orderly approach to many metabolic problems. The fetal liver is deficient in many substrates and enzymes. At birth it has approached a reasonable state of maturity except in certain rare "inborn errors of metabolism." Galactosemia appears to be caused by a deficiency of phosphogalactose transferase, an enzyme which catalyzes the reaction between galactose-1-phosphate and uridine-diphosphate-glucose to form uridine-diphosphate-galactose and glucose-1-phosphate. In some instances, the deficiency is questionably transient or may be relieved by alternative metabolic pathways.[20] Glycogen storage disease (von Gierke's disease) appears to be caused by a deficiency or absence of the enzyme glucose 6-phosphatase or in some other forms of the disease, by certain abnormalities in the structure of glycogen caused by other enzymes.

Glucose, galactose and levulose tolerance tests are grouped as liver function tests. However, the hepatic reserve permits rapid readjustments to certain pathologic changes without affecting the results of these tests. These reservations, coupled with the enormous extra-hepatic influences and the inter-relationships of the three chief pathways of metabolism, lessen the reliability of such tolerance tests for hepatic study.

The Metabolism of Fats

The analysis of serum components of lipids is rarely carried out in the evaluation of jaundice. The liver's capacity for the synthesis and degradation of fat is as profound as that for carbohydrate metabolism, if not more so. Serum cholesterol may well be within normal limits despite massive changes in the liver. Cholesterol is lowest in the first few days of life, chiefly due to the lack of enzymatic synthesis of this compound from two carbon units.[21] On the other hand, cholesterol metabolism may introduce other extrahepatic influences. We are aware of other disease entities producing increases in cholesterol levels in serum.

Many contributors to this field accept the cholesterol–cholesterol ester ratio as a potential indicator of hepatic disease. Cholesterol is increased in obstructive forms of jaundice and decreased in extensive parenchymal involvement. It is rarely affected by physiologic hyperbilirubinemia. A ratio

[15] Moore, D. H., Du Pan, R. M., and Buxton, C. L.: An electrophoretic study of maternal, fetal and infant sera, Am. J. Obst. 57:512 (Feb.) 1949.

[16] Oberman, J. W., Gregory, K. O., Burke, F. G., Ross, S., and Rice, E. C.: Electrophoretic analysis of serum proteins in infants and children. I. Normal values from birth to adolescence, New Eng. J. Med. 255:743 (Oct. 18) 1956. Oberman.[17]

[17] Oberman, J. W., Gregory, K. O., Burke, F. G., Ross, S., and Rice, E. C.: Electrophoretic analysis of serum proteins in infants and children. II. Serum gamma-globulin levels in selected infections and "hypersensitivity" diseases in childhood, New Eng. J. Med. 259:855 (Oct. 30) 1958.

[18] Popper, H., Huerga, J., Steigmann, F., and Slodski, M.: Turbidimetric gamma-globulin determinations in hepatobiliary diseases, J. Lab. & Clin. Med. 35:391 (1950).

[19] Salmon, G. W., and Richman, E. E.: Liver function in the newborn period, J. Pediat. 23:522 (Nov.) 1943.

[20] Isselbacher, K. J.: Evidence for an accessory pathway of galactose metabolism in mammalian liver, Science 126:652 (Oct. 4) 1957.

[21] Bogin, M., Gottfried, S. P., Levysky, N. V., and Graf, C.: Serum bilirubin levels in the newborn infant and their relation to clinical icterus and liver function studies, J. Pediat. 53:529 (Nov.) 1958.

of 50 per cent or less is an indication of liver involvement. If the ratio drops rapidly in hepatitis, an ominous prognosis is predicted. Because of many confusing factors influencing the results, this test is not commonly used as a hepatic function test.

Miscellaneous

Enzymes

The alkaline phosphatase level may indicate adult obstructive hepatic disease. It is of limited value in obstructive disease of infancy, frequently being normal even in congenital biliary atresia.[12] The Kupffer cells, the reticulum lining of the sinusoids, and the bile canaliculi are the chief sites of excretion of this enzyme. Hepatic cells are devoid of this function.

In children it is elevated in infectious hepatitis, often rising before any increase in bilirubin is noted. It may or may not be elevated in congenital biliary atresia. As jaundice begins to mount, we may find that alkaline phosphatase levels become disproportionate to the bilirubin levels.

In a recent work of Kove and others,[22] the serum transaminases have been investigated in the neonatal period (Table 3). The authors reported normal values in physiologic hyperbilirubinemia. They concluded that the serum bilirubin does not affect the activity of either the glutamic oxaloacetic transaminase or the glutamic pyruvic transaminase. In jaundice due to extrahepatic causes, they are within normal limits. Rise in activity may be seen in biliary atresia and inspissated bile syndrome. The activity increases slowly and progressively to levels of 300-800 units. In the latter, the initial activity is higher. In acute hepatitis there is a rapid rise during the stages of hyperbilirubinemia to values exceeding 800 units. This is followed by a sharp decline. Further studies on the uses of this laboratory test need to be done before it can be accepted as a routine hepatic function test. In addition, the outlook for other tests of enzyme activity in the liver is promising.

TABLE 3.—*Serum Transaminase Activity in the Neonatal Period (from Kove,* et al.)

	NUMBER OF PATIENTS	GO-T (UNITS/ML)	GP-T (UNITS/ML)
Normal (infants)	63	5-120	5-90
Normal (adults)	....	5-45	5-45
Physiologic hyperbilirubinemia	19	5-120	5-90
Neonatal hepatitis	1	Rapid rise to 800 or more	Rapid rise to 300 or more
Congenital biliary atresia	2	Slow rise to 300-800	Similar to GO-T
Inspissated bile syndrome	13	Slow rise to 300-800	Similar to GO-T
Hemolytic disease (Rh or ABO incompatibility)	7	5-120	5-90

Bromsulfalein Excretion Test (B.S.P.)

The glucuronide synthesis can involve certain foreign substances or exogenous dyes introduced into hepatic circulation. As mentioned earlier, the process has been shown to be functionally immature. Therefore, as a hepatic test involving this system, it can be expected to show an increased retention of the dye in neonatal life and yet not be indicative of any hepatic disease. In older infants and children it can be used as a test of "functioning hepatic mass."[10] A retention of at least 10-30 per cent at 45 minutes can be expected in the first week of life. Often times in chronic or long-standing hepatic diseases increased retention of the dye is seen. It is also prolonged in infectious hepatitis even when other tests have returned to normal. Certain "corrective factors" must be kept in mind. One must remember that fever and other "stress" factors vitiate the results and should be taken into account.

The examination for radioactive bromsulfalein in stools following intravenous injection of the dye has been described by Geppert and Brent. Most recently the same authors have introduced into pediatric diagnosis the so-called I^{131} rose bengal, given recognition by Israels and Israels.[24] The principle is similar to that of the radioactive bromsulfalein test. It is hoped to be of some value in the differentiation of biliary atresia from other forms of neonatal jaundice.

Needle Biopsy and Surgical Exploration

The clinical course of hepatic illnesses can be followed with periodic liver function tests. At times, however, the decision for surgical intervention may be preceded by a needle or open biopsy of the liver. The need for this procedure is still highly controversial.[25] There are some who feel that there is little or no correlation between functional derangement and histologic alterations. Therefore, there is a variable time in which one is inclined to delay surgical intervention. In a study of 37 infants, however, Christy and Boley[26] showed

[22] Kove, S., Goldstein, S., and Wroblewski, F.: Activity of glutamic oxaloacetic transaminase in the serum in the neonatal period, Pediatrics 20:584 (Oct.) 1957. Kove.[23]

[23] Kove, S., and Goldstein, S.: Serum transaminase activity in neonatal period. Valuable aid in differential diagnosis of jaundice in the newborn infant, J.A.M.A. 168:860 (Oct. 18) 1958.

[24] Israels, S., and Israels, L. G.: Newer laboratory procedures, their application to investigation of pediatric disorders, Ped. Clin. of N. Am. (May) 1958, p. 364.

[25] Zollinger, R. M., Goldblatt, H., Warren, K. W., Wheeler, W. E., and Rossmiller, H. R.: Jaundice: symposium, Postgrad. Med. 22:171 (Aug.) 1957. Christy.[26] Harris.[27] Harris.[28]

[26] Christy, R. A., and Boley, G. O.: The relation of hepatic fibrosis to concentration of bilirubin in the serum in congenital atresia of the biliary tract, Pediatrics 21:226 (Feb.) 1958.

conclusively the extent of liver fibrosis caused by persistently elevated bilirubin levels in order to determine the logical time to perform a surgical exploration. Since some infants showed hepatic fibrosis before the age of two months, they suggested early laparotomy, biopsy, or cholangiography (Swenson) followed by surgical correction of the amenable lesion if possible.

Most schools feel that needle biopsy should be performed. Complications such as perihepatitis, pneumothorax, hemorrhage, or bile peritonitis are relatively rare. Sherlock reported a mortality rate of 0.17 per cent.[29] Others have reported lower mortality rates.[25] The indications for surgical exploration are discussed by Hsia and Gellis[12] (Table 4).

TABLE 4.—*Indications for Surgical Exploration in Neonatal Jaundice (from Hsia and Gellis).*

1. Absence of bile in stools or duodenal secretion.
2. No urobilinogen in urine.
3. Initial bilirubin level low, followed by slow rise and no variability.
4. Flocculation tests negative.
5. No blood incompatibility.

Conclusions

In this brief review liver function tests in pediatrics were discussed in the light of present theories regarding hepatic metabolism.

[27] Harris, R. C.: Liver function tests in infancy, Bull. N.Y. Acad. Med. 28:721 (Nov.) 1952.
[28] Harris, R. C., Anderson, D. H., and Day, R. L.: Obstructive jaundice in infants with normal biliary tree, Pediatrics 13:293 (Apr.) 1954.
[29] Cited by Prior.[14]

Those tests which are applicable to pediatric practice were presented.

It is agreed that certain factors, which have to be borne in mind, influence the results of these tests. Specific examples have been cited; as for example, physiologic hyperbilirubinemia, familial nonhemolytic jaundice, galactosemia, and glycogen storage disease. These serve to illustrate the relative hepatic immaturity of enzymatic systems of the newborn and infant and how they influence the interpretations of liver function tests. Familiarity with the normal expectations for certain age groups, and the physiological basis, range of response and limitations of each test have been stressed.

Surgical intervention may become mandatory in prolonged obstructive jaundice and contraindicated in inspissated bile syndrome and other forms of medical jaundice. Liver biopsy and operative cholangiography with special reference to the time factor were discussed. In conclusion, liver function tests should be wisely selected after a critical assessment of the patient, and with the technical and theoretical knowledge of each test selected in mind.

Summario in Interlingua

Tests del function hepatic es discutite in relation a theorias physiologic e enzymatic del function hepatic. "Immaturitate hepatic" de neonatos es exemplificate in hyperbilirubinemia physiologic. Es discutite le evalutation de certe statos pathologic in que le hepate es interessate.

The AMEF will transmit your donation in full, in your name, to the medical school of your choice.

This mysterious and lethal lesion causes between one-fourth and one-third of neonatal deaths in Hawaii. Cesarean section predisposes to it, as does fetal distress.

Pulmonary Hyaline Membrane Disease In the Newborn

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ALTHOUGH the pulmonary hyaline membrane was first discovered by Hochheim of Berlin in 1903, it is only relatively recently we have learned to associate the entity with its dire significance clinically.

Babies suffering from this disorder usually do well for the first few hours of life, and then show signs of respiratory difficulty with sternal retractions, grunting respirations, cyanosis, apnea, and death in 24 to 48 hours.

Incidence

The incidence of pulmonary hyaline membrane disease as reported by different investigators has varied widely, and this is due to different methods of handling the newborns, various percentages of premature infants in their series, more extensive and complete examination of pathological specimens removed, and different interpretation by the investigators. Helwig[1] reviewed 159 autopsies of live-born infants; hyaline membrane disease was found on two occasions, giving a percentage of 1.3. In contrast to this study, Ahvenainen[2] reviewed 81 necropsied specimens and found the disease in 50 per cent. In our series, the total number with hyaline membrane disease was 82, or 28 per cent of 291 autopsies on live-born infants. Similar results were reported by Potter,[3] Bruns,[4] and Blystad.[5]

Miller and Jennison,[6] of Kansas City, reviewed in 1950, 4,117 consecutive autopsies on infants weighing over 1,000 grams at birth and dying during the first 48 hours of life. Sixty-five per cent had hyaline-like membranes in the lungs.

DR. HARBINSON

It was routine, at this time, to administer 100 per cent oxygen during the first 24 hours of life.

In 1952, Blystad, Landing, and Smith,[5] of Boston, in a review of 322 consecutive postmortems performed between 1941 and 1948 on full-term babies living at birth, and later posted, found 22 per cent had hyaline membranes.

One-third of those delivered by cesarean section had hyaline membranes.

Forty-three per cent of complications in the group with hyaline membranes were formed by cesarean section, placenta previa, and fetal distress.

Claireaux,[7] of London, England, found hyaline membrane disease at autopsy in 108 out of 376 live-born infants coming to autopsy between 1948 and 1952. Of these 76 were males and 32 females. Eighty per cent occurred in prematures and 66 per cent died during the first 24 hours.

Hyaline membraine disease was the sole pathological lesion in 75 per cent. Of those found to have hyaline membranes, 20 per cent were delivered by cesarean section, and of these, 59 per cent were because of pre-eclampsia.

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[1] Helwig, F. C.: Congenital aspiration pneumonia in stillborn and newborn infants. Analysis of 159 necropsy examination, Am. J. Obs. & Gyn. 26:849 (Dec.) 1933.

[2] Ahvenainen, E. L.: On membranes ("Vernix Membranes") caused by amniotic fluid in lungs of newborn and on interstitial emphysema as well as spontaneous pneumothorax, Ann. Med. Int. Fenniae. 36:367 (Fasc. 3) 1947.

[3] Potter, E. L.: Pathology of prematurity, J. of the Am. Med. Women's Assoc. 5:391 (Oct.) 1950.

[4] Bruns, P. D., Shields, L. V.: The pathogenesis and relationship of the hyaline-like pulmonary membrane to premature neonatal mortality, Am. J. of Obs. & Gyn. 61:953 (May) 1951.

[5] Blystad, W., Landing, B. H., and Smith, C. A.: Pulmonary hyaline membranes in newborn infants. Statistical morphologic and experimental study of their nature, occurrence and significance, Ped. 8:5 (July) 1951.

[6] Miller, H. C., and Jennison, M. H.: Pulmonary hyaline-like material in 4,117 consecutive births, incidence, pathogenesis, and diagnosis, Ped. 5:7 (Jan.) 1950.

[7] Claireaux, A. E.: Hyaline-membrane in the neonatal lung, Lancet II:15 (Oct. 10) 1953.

TABLE 1.—*Comparison of the Incidence of Hyaline Membrane Disease between Kapiolani Hospital and St. Francis Hospital.*

	KAPIOLANI HOSPITAL			ST. FRANCIS HOSPITAL		
	No. of Autopsies	*No. of H.M.D.*	*Incidence of H.M.D.*	*No. of Autopsies*	*No. of H.M.D.*	*Incidence of H.M.D.*
Stillborns	77	0	0%	56	0	0%
Live-births	196	47	24%	95	35	37%
Full-term	52	5	10%	18	4	22%
Premature	144	42	29%	77	31	40%

H.M.D. = Hyaline Membrane Disease.

TABLE 2.—*Hyaline Membrane Disease According to the Birth Weight.*

	BIRTH WEIGHT IN GRAMS					
	500-1,000	1,000-1,500	1,500-2,000	2,000-2,500	MORE THAN 2,500	TOTAL
St. Francis Hospital	5	14	7	5	4	35
Kapiolani Hospital	2	19	14	7	5	47
Total	7	33	21	12	9	82
Approximate percentage with hyaline membrane disease	9	40	26	15	11	

Incidence in Hawaii

The following analysis comprises 82 cases of hyaline membrane disease detected in 424 consecutive autopsies performed at the St. Francis Hospital and Kapiolani Maternity Gynecological Hospital from January, 1953, to June, 1958, on infants dying in the neonatal period. Table 1 illustrates number of live births, stillborn, full-term, and premature deliveries, together with the incidence of hyaline membrane disease.

Table 2 shows the distribution of the cases according to the birth weight. As reviewed by Tran-Dinh-De and G. W. Anderson,[8] the highest incidence is observed among infants weighing from 1,000 to 2,500 grams. In our survey approximately 40 per cent were between 1,000 and 1,500 grams and 26 per cent among the group of 1,500 to 2,000 grams. Fifty cases (61 per cent) occurred in males and 32 (39 per cent) in females. When the birth weight is the same, female infants are more mature than males and the former probably survive hyaline membrane better than the latter.

Table 3 shows length of survival time after birth. All but one patient died within 72 hours. The single patient who lived for 100 hours 25 minutes had associated anomalies of ventricular septal defect, cleft palate, and pourchette of vulva into the anus. The infant who survived the shortest interval of time weighed 1,400 grams at birth and expired two hours nine minutes later. The shortest time interval from birth to death was one hour according to the report of Blystad *et al.*,[5] but Miller[9] reported hyaline membrane disease among stillborn infants. Out of 133 consecutive postmortem examinations of stillborn infants, there was no hyaline membrane disease in our series.

Table 4 demonstrates the relationship of incidence of hyaline membrane disease to birth weight and length of survival time. There is a significant rise in death rate six to twelve hours after birth in infants weighing 1,000-1,500 grams. In an infant weighing less than 1,000 grams at birth, death within 24 hours is the usual occurrence. For infants weighing 1,000-2,500 grams at birth, the first 48 hours is considered the critical period.

As shown in Table 5, there were nine patients with hyaline membrane disease whose birth weight exceeded 2,500 grams. Of these, four were delivered by cesarean section, three being repeat sections.

The above surveys indicate that hyaline membrane disease occurs more frequently in prematures, and more often in males. There is a higher incidence in those delivered by cesarean section, and where there is associated fetal distress; e.g., placenta previa and pre-eclampsia.

The Nature of the Membrane

According to Miller and Hamilton,[9] the amount of hyaline membranes in the lungs is definitely related to the birth weight. They found the incidence of positive fat stains opposed the dis-

[8] Tran-Dinh-De, and Anderson, G. W.: Hyaline-membranes associated with diseases of the newborn lungs. A review of the literature. Obs. & Gyn. Survey 8:1 (Feb.) 1953.

[9] Miller, H. C., and Hamilton, T. R.: Pathogenesis of the "vernix-membrane": relation to aspiration pneumonia in stillborn and newborn infants. Ped. 3:735 (June) 1949.

TABLE 3.—*Length of Survival (Hours).*

Hours after delivery	BIRTH–6	12	18	24	36	48	72 AND OVER	
No. of cases	9	21	12	10	13	7	9	1

Deaths Occurred During the:	*Number*	*Per Cent*
1st day	52	63
2d day	20	24
3d day	9	11
Thereafter	1	1

TABLE 4.—*Relationship between the Birth Weight, Length of Survival Time, and Occurrence of Hyaline Membrane Disease.*

BIRTH WEIGHT										
4,000	....	....	....	....	....	....	....	....	....	....
3,500	....	....	....	....	1	....	....	....	....	1
3,000	....	1	....	1	....	....	1	....	....	3
2,500	....	....	2	....	1	....	2	....	....	5
2,000	....	3	1	2	2	3	1	....	....	12
1,500	3	2	2	3	4	2	3	1	1	21
1,000	4	11	6	4	5	2	1	....	....	33
500	2	4	1	....	....	....	....	....	....	7
Total	9	21	12	10	13	7	8	1	1	82

0 6 12 18 24 36 48 60 72
Survival time (hours)

TABLE 5.—*Cases of Hyaline Membrane Disease Whose Birth Weight Exceeded 2,500 Grams.*

CASE NO.	BIRTH WEIGHT	OBSTETRICAL INFORMATION
A-98-53	3040 grams	Double footling
A-1-55	3150 grams	Cesarian section
A-53-55	2573 grams	Normal spontaneous delivery with episiotomy
A-115-55	2517 grams	Premature rupture of membrane
KA-8-54	2890 grams	Normal spontaneous delivery
KA-19-54	3087 grams	Repeat cesarian section
KA-15-57	3640 grams	Repeat cesarian section
KA-31-57	2880 grams	Repeat cesarian section
KA-13-58	2500 grams	Low forceps delivery

tribution of hyaline-like membranes in respect to time of death and birth weight. This notable absence of fat from hyaline-like membranes suggests the operation of some other factor than aspiration of vernix, e.g., injury to the lung.

Blystad *et al.*,[5] from experimental work on animals, feel that if sufficient amniotic fluid is introduced into the trachea, and if respirations are continued for a sufficient length of time (at least one hour) membranes may be produced. However, since the protein content of the amniotic fluid is relatively low, considerable water must be removed in order to produce the concentration in these dense homogeneous membranes.

Ranström[10] feels if membranes are formed from aspirated material from which water has been absorbed, this material must have prevented gaseous exchange from the very start—before membrane formation.

If membranes are formed from capillary exudation—this may be the final obstacle to gaseous exchange—atelectasis is still the most important factor. "Membrane asphyxia" must be reserved for those cases with widespread membrane formation and secondary atelectasis.

Claireaux[7] found the period acid–Schiff stain weakly positive, indicating polysaccharide, and often neutral fat could be detected. By taking sufficient blocks, he was able to show membranes are formed from individual squames. By centrifuging amniotic fluid and incubating the cells, he found they became acidophilic, lost their nuclei, and coalesced to form hyaline masses.

Wagner,[11] applying histochemical techniques, found the hyaline membranes contained mucoproteins or glyco-proteins. Examination of amniotic fluid aspirated from the uterus at the time of elective cesarean section revealed its mucoprotein content to be 80 mgm per cent and polysaccharide content 11 mgm per cent. He postulated the membrane is derived from endogenous mucus from the nasopharynx, buccal cavity, and the bronchial tree.

Etiology and Pathogenesis

Craig, *et al.*[12] have shown by injection of gelatin–India ink and latex masses under controlled conditions into the lungs of infants with asphyxia associated with hyaline membrane formation, that such membranes prevent the filling of distal alveoli. They postulate that these membranes obstruct the flow of air to the distal alveoli and prevent their expansion, thus decreasing the compliance of the lung.

Briggs and Hogg[13] believe that hyaline-like atelectasis, hyaline membrane, and pulmonary hemorrhage appear to be closely interrelated in time and pathogenesis.

[10] Ranström, S.: On the effect of the hyaline membranes in the lungs of newborn infants, Acta Paediat. 42:323 (July) 1953.

[11] Wagner, J. C.: Histochemistry of pulmonary hyaline membrane in newborn infants and its interpretation, Lancet II:13 (Sept. 25) 1954.

[12] Craig, J. M., Fenton, K., Gitlin, D.: Obstructive factors in the pulmonary hyaline membrane syndrome, Ped. 22:847 (Nov.) 1958.

[13] Briggs, J. N., and Hogg, G.: Perinatal pulmonary pathology, Ped. 22:41:Part I (July) 1958.

The experimental approach has been in two main directions: first, an attempt to show the role of aspiration of amniotic sac content and, second, the production of membranes following damage to the lung or pulmonary circulation.

A similar membrane is found in such conditions as rheumatic pneumonitis, tuberculosis, radiation pneumonitis, nitrous gas exposure, uremia, and viral pneumonitis.

Berfenstam[14] has succeeded in producing hyaline-like membranes in oxygen-intoxicated rats. An atmosphere of 90 to 100 per cent oxygen is required for at least 60 to 70 hours for the production of membrane in the animal lung. It is unlikely that any infant will be exposed to 100 per cent oxygen for very long.

This same worker[15] has studied ciliary activity in oxygen-intoxicated rabbits and found disturbance of activity. He concludes that if this can be applied to newborn prematures, oxygen therapy ought to be investigated to insure that the concentration used is not producing ciliary stasis and thereby increasing the tendency to form hyaline membrane.

Attention is therefore directed to a fundamental disorder of the pulmonary circulation, for hyaline membrane is almost invariably associated with pulmonary edema, severe pulmonary congestion, and intra-alveolar hemorrhage.

Lynch and Cameron[16] experimentally produced membranes in animals and tried to correlate serial EKG findings. Tracings suggested left ventricular preponderance, and they portend left heart failure with pulmonary edema as the disease progresses. They believe left ventricular strain leads to elevation of serum potassium and phosphate with consequent lowering of the pH and glycogen depletion of the myocardium.

Lendrum[17] feels hyaline membrane is a manifestation of left heart failure—the cardiovascular system is not yet ready to make adjustment to extrauterine life and failure to make an adjustment imposes an excessive load on a barely marginal pulmonary vascular bed. Resorption of edema fluid will exceed transudation from the capillaries when the pressure in the pulmonary vessels drops sufficiently.

Arey believes vascular damage or increased capillary fragility is the common factor responsible for hyaline membrane disease. Anoxia with increased capillary damage may lead both to aspiration of excess amounts of amniotic debris and to the formation of pulmonary hyaline membranes.

We need to learn more about the state of the pulmonary circulation in the newborn, and the response of the pulmonary vessels to the stress of extrauterine existence.

Whether the pulmonary hyaline membrane is due to left ventricular failure secondary to an immature cardiovascular system, or to some substance increasing capillary permeability, or both, at any rate it is composed of exudate from the pulmonary capillary bed. This organizes and contains a large amount of fibrin. It would appear that the membrane, once formed, prevents flow of air to the distal alveoli, resulting in atelectasis.

Therapy

The cause being unknown, treatment is empirical. The following may, or may not be used:

1. Aspiration of the stomach in babies delivered by cesarean section
2. 35-40 per cent oxygen mist
3. Antibiotics
4. Digitalization
5. Steroids

The prophylactic treatment of this disease cannot be emphasized too highly: (1) Adequate prenatal care including good control of the diabetic mother. (2) A critical assessment of the need for cesarean section; we contend that the dictum "once a section, always a section" no longer necessarily holds. If delivery by section is indicated, it should be performed, whenever possible, when the patient goes into labor.

Case Report

Abstracted below is the case report of an infant, who recently died of this disease under our observation.

A premature male infant was born by low-transverse cesarean section, done because of previous section in 1957 due to pre-eclampsia and fetal distress.

The baby weighed 5 lbs., 6½ oz. and was 18½ inches in length. He breathed spontaneously, although subcostal retractions were noted a few minutes following delivery. About ten cc of liquid resembling amniotic fluid were aspirated from the stomach and the baby was placed in 40 per cent oxygen mist.

The mother was a 19-year-old Hawaiian-Caucasian, gravida III, para II, who had inactive rheumatic heart disease with mitral insufficiency. In addition, she had undergone in 1957 removal of the thyroid with the exception of a portion of the right lobe, along with radical dissection of the left neck, for carcinoma of the thyroid.

A clinical diagnosis of hyaline membrane disease seemed definite about 3 P.M.—some five hours after birth—and the baby was treated accordingly with chloramphenicol, digitoxin, and cortisone. The diagnosis was further substantiated by x-ray, which showed diffuse granularity in both lung fields (Fig. 1). The baby expired 19 hours after birth.

[14] Berfenstam, R., Edlund, T., and Zettergren, C.: The hyaline membrane disease. A review of earlier clinical and experimental findings and some studies on the pathogenesis of hyaline membranes in oxygen-intoxicated rabbits, Acta Paed. 47:82 (Jan.) 1958.

[15] *Ibid.*: The influence of high oxygen concentration on ciliary activity in the respiratory tract. An experimental study on rabbits, Acta Paed. 47:527 (Sept.) 1958.

[16] Lynch, M. G., and Cameron, G. C.: Hyaline membrane disease with EKG comment, Ped. 48:602 (May) 1956.

[17] Lendrum, F. C.: The pulmonary hyaline membrane as a manifestation of heart failure in the newborn infant, J. Ped. 47:149 (Aug.) 1955.

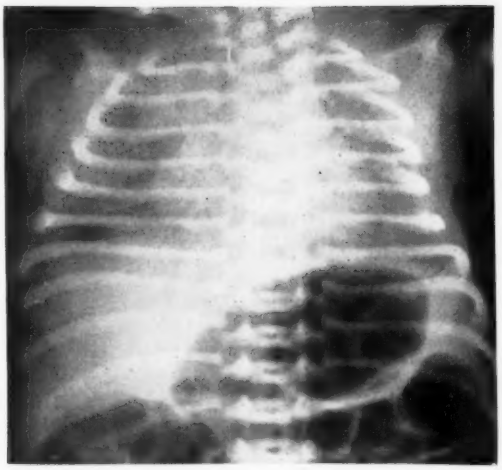

FIG. 1.—Portable x-ray of chest taken five and a half hours after delivery, showing thymus shadow in right upper lung field and plate atelectasis in the right lower lung field. Diffuse granularity is seen in both lung fields indicating scattered atelectasis. Air-containing bronchi suggest partial atelectasis.

findings of the disease found at postmortem are related.

Recent experimental work suggests that the disorder is due to some insult to the integrity of the pulmonary capillary wall.

Empirical therapy is related, with particular emphasis on prophylaxis.

Summario in Interlingua

In duo hospitales de Honolulu, 424 necropsias consecutive, effectuate inter 1953 e 1958 in infantes morte-nate o moriente in le periodo neonatal, includeva 82 casos del syndrome de membrana hyalin pulmonar. Omne iste casos concerneva infantes vive-nate. Le proportion de iste casos in le total de necropsiate infantes vive-nate esseva 24 pro cento in un hospital e 37 pro cento in le altere. Un medietate del casos concerneva infantes de pesos natal de minus que 1.500 grammas. Tres quartos del infantes in iste gruppo pesava minus que 2.000 grammas. Duo tertios moriva durante le prime die de lor vitas e un quarto additional durante le secunde die. Ex le total de nove vic-

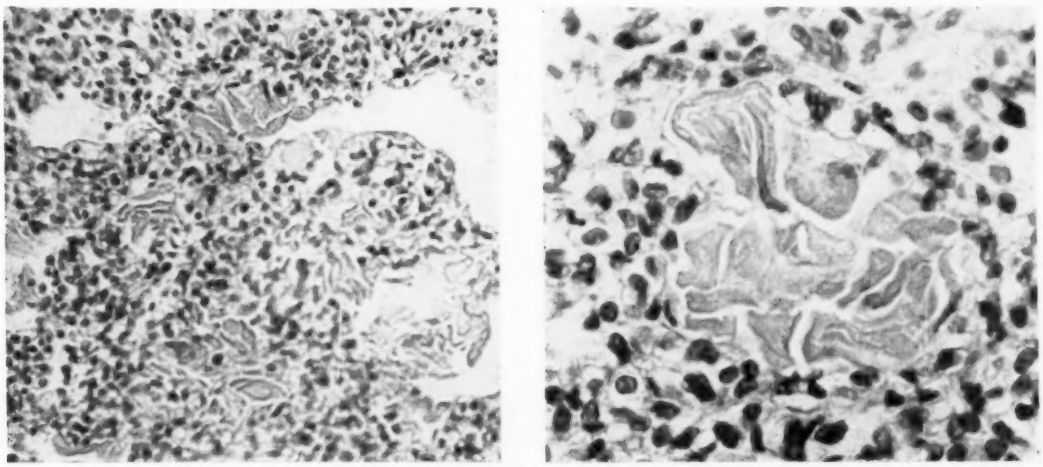

FIG. 2(a).—Low-power view of lung showing membrane lining alveoli with areas of atelectasis between affected alveoli. (b). High-power view of same.

At autopsy, this baby showed the usual findings of the pulmonary hyaline membrane; namely, lungs which are pink-red and firm. Microscopically, many alveoli were found to be lined with dense, homogeneous pink membranes with zones of atelectasis between them (Fig. 2).

Summary

The case report of a premature male infant delivered by cesarean section is presented to illustrate the typical clinical course in hyaline membrane disease; in spite of all known forms of therapy, the infant succumbed, and the typical

timas del syndrome qui habeva un peso natal de plus que 2,500 grammas, quatro esseva parturite per section cesaree, un per forcipe de sortita, un in presentation de pedes, e duo de modo spontanee normal.

Le causa del phenomeno non es cognoscite, sed on suspice lesion de pariete capillar.

The authors wish to thank Drs. Raid Chappell and Grant Stemmermann, pathologists at St. Francis and Kuakini Hospitals respectively, for their invaluable assistance.

Anemia of the newborn can result from bleeding into the maternal circulation.

Fetomaternal Transfusion Syndrome

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FETOMATERNAL TRANSFUSION as a cause of severe anemia in a newborn has been only recently recognized. It is now clear that antepartal passage of fetal blood into the maternal circulation can happen. It is strange that this condition has remained so long unrecognized.

It was in 1948 that Wiener[1] first suggested that one of the causes of newborn anemia was occult bleeding of the fetus across the placental barrier into the mother. Adequate confirmatory studies to support this viewpoint were not realized until several years thereafter. However, since then, there have been numerous other documented cases[2] to firmly establish this disease as a real entity.

In 1955 Rucknagel and Chernoff[5] reported finding fetal hemoglobin in blood of pregnant women, and demonstrated that levels above 2 per cent were never attained. This finding of levels of fetal hemoglobin below 2 per cent in normal pregnancies thus provides a simple test to exclude the diagnosis, any level above 2 per cent being significant and indicating that the blood of the fetus has escaped into the bloodstream of the mother.

Basing our case on this laboratory examination and the rather typical clinical picture, we would like to add the following case report to the literature. We believe that more practicing physicians should be alerted as to the recognition of this disease, and also as to the relative ease of detection of these cases.

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† Department of Pediatrics, Straub Clinic.

[1] Wiener, A. S.: Diagnosis and treatment of anemia of newborn caused by occult placental hemorrhage, Am. J. Obst. and Gyn. 56:717 (Oct.) 1948.

[2] Chown, B.: Anemia from bleeding of fetus into mother's circulation, Lancet 1:1213 (June 12) 1954. Chown.[3] Wickster.[4]

[3] Idem: Fetus can bleed: 3 clinicopathological pictures, Am. J. Obs. and Gyn. 70:1298 (Dec.) 1955.

[4] Wickster, G. Z.: Posthemorrhagic shock in the newborn, Am. J. Obs. and Gyn. 63:524 (Mar.) 1952.

[5] Rucknagel, D. C., and Chernoff, A. I.: I. Immunologic studies of pregnant woman. III. Fetal hemoglobin changes in circulation of pregnant women, Blood 10:1092 (Nov.) 1955.

Case Report

A 3.2 kg Caucasian male infant was delivered at Wahiawa General Hospital at 5 A.M. on January 9, 1958. He was the first product of conception for this 31-year-old mother, and the entire pregnancy was rather uneventful, except for mild hypertension, which was adequately controlled by reserpine. There was a question of the infant's being two weeks overdue, but both labor and delivery were reported to be normal. No note of any fetal distress was made, although the amniotic fluid was reported to be grossly contaminated with meconium at the time of delivery. When the fetus was delivered, he appeared pale and very listless and respirations were hard to start.

In view of this serious picture, a complete blood count was done immediately, revealing an erythrocyte count of 1.43 million and a hemoglobin level of 5.6 gm. He was immediately transferred to Kauikeolani Children's Hospital for further evaluation and management.

Examination on admission (at approximately 1½ hours of age) revealed a well-developed full-term infant, who appeared rather active. He had a good cry and the Moro and sucking reflexes were present. The only abnormal finding was the marked pallor.

A repeat blood count showed an erythrocyte count of 1.16 million with a hemoglobin of 4.8 gm. The total corrected leukocyte count was 23,935 with 32 per cent neutrophils, 13 per cent stabs, 53 per cent lymphocytes, and 2 per cent monocytes. Marked anisocytosis and hypochromia were noted, as well as 41 nucleated erythrocytes per 100 leukocytes. Both the mother's and the infant's blood were type O Rh negative, and the Coombs' test, as well as tests for other irregular antibodies, were negative. The bilirubin level of the infant's serum was 1 mg per ml.

Because of the marked anemia, a transfusion of 50 cc of whole blood was given. He tolerated the entire procedure very well, and the hemoglobin level was raised to 8.9 gm. However, that evening, about 12 hours after admission to this hospital, the infant was noted to have a rectal temperature of 94° F. although he appeared to be clinically normal. He was transferred to another incubator, where a higher atmospheric temperature was set, and the following morning, he was noted to have a fever of 101° and appeared spastic. The Moro reflex seemed only fair, the abdomen was tense and distended, and grunting respirations were noted. He had also vomited his initial glucose water feedings.

The picture of brain damage became even more evi-

dent later that day, with marked opisthotonos, spastic extremities, and complete loss of the Moro reflex. Another 50 cc of whole blood were again administered at this time with no untoward reaction. That evening the skin began to appear waxy, and pitting edema was noted on the back. Urine and meconium were being passed by this time.

Penicillin and streptomycin had been started on admission and were continued throughout hospitalization. All oral feedings were stopped in view of the marked edema. The opisthotonos persisted and a high-pitched shrill cry with grunting, jerky-type respirations was noted. Because of a markedly diminished urinary output at this time, 75 cc of 10 per cent glucose in distilled water were given intravenously twice a day to maintain nutrition and supply fluids until normal function could be reestablished.

On the third hospital day it was evident that the edema was less pronounced and he had lost 150 gm of weight, having gained a total of 270 gm prior to this time since admission. He gradually improved, slowly losing all evidence of central nervous system involvement. By the eighth hospital day the sucking and Moro reflexes had completely returned. A voided urinalysis revealed 10-20 white cells per high power field at this time, and although he was asymptomatic, the other antibacterial agents were discontinued, and sulfisoxazole (Gantrisin) was started. He made a rather uneventful recovery from this point on, and weighed 3.6 kg at the time of discharge, appearing perfectly normal for a newborn.

At the time of discharge hemoglobin was 11.8 gm and the erythrocyte count 4 million. A voided urine was normal.

Most significantly, the specimen of blood drawn from the mother two days after her delivery of this infant revealed the presence of a fetal hemoglobin concentration of 6 per cent. This determination confirmed our clinical suspicions of a fetomaternal transfusion phenomenon being responsible for the severe anemia of this infant at the time of birth.

Subsequent followup in the office revealed a completely well infant, growing and developing normally. On April 10, 1958, when last seen at the office, he weighed 7.2 kg and the erythrocyte count was 3.9 million with a hemoglobin of 10.3 gm. Voided urine was again normal. Unfortunately, he left the Islands at the age of three months when his father was transferred from here, but additional followup correspondence from the parents at 11 months of age seems to indicate no sequelae from the disease process noted at delivery. He is growing well, is walking with assistance, and has a vocabulary of several words.

Discussion

The above case well illustrates the problem of a severe anemia in a newborn infant. It was obvious at birth that the infant was extremely pale and that there had been distress both before and after delivery as evidenced by the meconium staining of the amniotic fluid and the delayed onset of respirations at the time of delivery. A blood count immediately revealed the cause of the difficulties, and no time was wasted in referring this problem into Children's Hospital for more specific diagnostic studies and management. It is conceivable that this delay, necessitated by the transfer of the case from one hospital to another, might even be hazardous for such an infant suffering from posthemorrhagic shock, and an immediate blood transfusion of available compatible blood might have to be done in the delivery room.[6]

Recognizing that one of the causes of this severe anemia in our infant may have been due to a fetomaternal transfusion, an effort to confirm this impression was made. A specimen of maternal blood was obtained, and the amount of fetal hemoglobin in it was determined by the alkali denaturation method of Singer.[7] The finding of 6 per cent fetal hemoglobin level in the maternal circulation exceeded the maximum level of 2 per cent as demonstrated by the studies of Rucknagel and Chernoff on normal pregnant women, and this aided in establishing the diagnosis.

The management of posthemorrhagic anemia in a newborn is rather simple, once the diagnosis is made. Depending upon the severity of the disease process, transfusions of available compatible blood should be curative. Such infants should be carefully followed up to see if an iron deficiency type of anemia develops. Iron medication or further transfusions may then be indicated.[6]

Summary

One of the causes of severe anemia in the newborn is fetal bleeding across the placental barrier into the maternal circulation. This condition should be suspected in any newborn infant who appears very pale and may even be in a state of shock, manifesting both intra- and extrauterine distress such as meconium staining of the amniotic fluid, or difficulty in resuscitation.

Confirmation of this diagnosis entails serial determinations of the maternal blood for abnormal amounts of fetal hemoglobin, and differential agglutination studies. A positive indirect Coombs' test would also aid in the diagnosis.

Treatment consists merely of transfusions of an adequate amount of compatible blood. These infants should also be closely followed to note whether or not they develop a significant degree of iron deficiency type of anemia.

Summario in Interlingua

Anemia fetal pote resultar ab sanguination a in le circulation materne. Es reportate un caso de iste phenomeno. Le characteristica pathognomonic in iste caso es un contento de hemoglobina fetal in le sanguine materne de plus que le maximo normal de 2%. Transfusiones es curative.

[6] McGovern, J. J., Driscoll, R., DuToit, C. H., Grove-Rasmussen, M., and Bedell, R. F.: Iron deficiency anemia resulting from fetomaternal transfusion, New Eng. J. Med. 258:1149 (June 5) 1958.

[7] Singer, K., Chernoff, A. I., and Singer, L.: Studies in abnormal hemoglobins: their demonstration in sickle cell anemia and other hematologic disorders by means of alkali denaturation, Blood 6:413 (May) 1951.

The President's Page

WILLIAM N. BERGIN, M.D.

Writing this as I am on the first day of the year of the Pig, I feel that it is only appropriate that the special thanks of the Association should be expressed to Dr. Min Hin Li, who, since its inception, has been our Association's Chairman of the American Medical Education Foundation Committee.

As nearly as can be determined, our contributions through the AMEF during the first nine months of 1958 have averaged less than $4.00 a member, with no contributions from the outside islands. Just to prove that we have not seen the last of the big spenders, however, one Honolulu County member recently contributed $500.00—the largest single donation Dr. Li has received in all of his seven year tenure.

I am sure that many of us have contributed directly, so that these figures are not what they appear to be. The problem lies, I am sure, in the fact that each of us has a father-image of Diamond Jim Brady, and that some day we are really going to give a walloping donation to our respective schools. It is going to be a long time before any one of us suddenly finds himself in a position to give our schools a Brady-like gift, so we may as well lower our sights in the meantime and make our own "louse" books. Have your secretary make out twelve checks in the amount of $5.00 each, post-dating each check, for each month of the year. All you will have to do is sign your name twelve times and forward the checks on to Dr. Li, in care of the HMA. It amounts to only sixty bucks, but it is $56.00 more than we now seem to be averaging, and twelve post-dated checks are a helluva lot better than a pledge card.

* * *

His Excellency the Governor William F. Quinn has recently asked your President to serve on his Council on Education Beyond the High School in Hawaii. We succeed the Governor's committee headed by Mr. Malcolm MacNaughton, and they have provided us with a startling and somewhat alarming ten-year projection of the problem we in the islands must face by 1968. Their conservative estimate is that there will be double the number of students, requiring double the number of teachers and double the physical area of instruction. Copies of this report are available in our own library, as well as the public libraries, and it is my hope that our JOURNAL will soon carry portions, if not all, of this survey.

It is also the Governor's hope that the coming Legislature will recommend that the Territory become part of the Western Interstate Compact of Colleges, which would make available to local students the professional schools of eleven Western states with in-state tuition rates, the Territory picking up the tab for the difference. The proposition is so practical that it demands our Association's wholehearted support.

Kung Hee Fat Choy!

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[EDITORIALS]

Kauikeolani Hospital Jubilee

And ye shall hallow the fiftieth year . . .
it shall be a jubilee unto you.
LEVITICUS 25:10

The completion of a half century of operation of the Kauikeolani Children's Hospital is the occasion for this, the second Children's Hospital issue of the HAWAII MEDICAL JOURNAL. Their distinguished present Visiting Professor, Dr. Paul Gyorgy, reviews the hospital's postgraduate educational program in a guest editorial elsewhere in this issue. All the articles were contributed by the hospital's resident staff.

A four-day program to celebrate the occasion will begin on Sunday, April 19, at the hospital, at 2 P.M. There will be medical presentations on Monday and Tuesday at the hospital and at the Princess Kaiulani Hotel. Distinguished visiting participants will be Drs. Tague Chisolm and James Roswell Gallagher, from the University of Minnesota and Boston, respectively.

As we go to press, announcement has just been made of the departure of the hospital's Director of Education, Dr. Donald F. B. Char. He has accepted a pediatric research post at the University of Washington Medical School, in Seattle. His successor has not been announced.

The descendants of Emma Kauikeolani (Mrs. A. S.) Wilcox, Kauai matriarch whose memory the hospital perpetuates, may take great pride in this vigorous, progressive institution of healing and education. It is a credit to the Wilcox family, and to the entire community. We salute it, in this, its year of jubilee.

"Operation Hypo"

In 1957, at an invitational public meeting sponsored by the Honolulu County Medical Society and the National Foundation (for, at that time, Infantile Paralysis), the Medical Society was asked to organize a campaign to promote vaccination against poliomyelitis. The National Foundation offered a $14,000 grant to finance the program. The medical society's Public Service Committee, of which Dr. Masato Hasegawa was chairman, was given the task. A representative Executive Committee was organized and a professional public relations consultant employed, and on October 1, 1957, the campaign began.

The initial period—"Phase I"—was devoted to an educational effort, urging everyone to "Get Your Polio Shots NOW—See Your Doctor." Physicians agreed to accept a fee of not over $2 per inoculation—well under the cost. An estimated 39,000 injections were given during Phase I.

Phase II, involving group inoculations for employees, club members, etc., given on their own premises, ran from November 15 through February 28 for first and second inoculations. In 1958 Dr. R. Varian Sloan took over as chairman of the Public Service Committee and superintended the operation. Nearly two hundred physicians donated

their services for these group inoculations, many of them conducting as many as 15 or 18 sessions; one, Dr. John C. Milnor, was awarded a plaque in recognition of his having conducted 25. Almost 50,000 injections were given in Phase II. Phase II was actually completed (with the third dose of vaccine) on September 30.

Phase III began March 4, 1958, with the scheduling of Community Vaccination Centers in almost every elementary school district on Oahu. May 13 marked the completion of the first two doses, and by December 18, children had been given nearly 25,000 doses of vaccine. In addition, during the summer, 2,696 emergency injections were given to help stem an increase of polio cases noted at that time.

Of the grand total—an estimated 116,000 injections—only 8,058 were given without charge; the remainder were paid for, in whatever amount (up to $2) the patient felt able to afford. The total sum realized still awaits auditing, but it will exceed $30,000, and this amount has already been designated by the Board of Governors of the medical society as a donation from Operation Hypo to the building fund of the Honolulu County Medical Library.

The entire operation—though somewhat disappointing in its results so far as immunization of the entire community against poliomyelitis is concerned—reflects great credit on the community committee which organized it under the aegis of the Honolulu County Medical Society's Public Service Committee, and on Dr. Hasegawa and Dr. Varian Sloan, who bore the brunt of the hard job of keeping the project moving. It would reflect special credit on the physicians who gave so freely of their time and efforts to perform the inoculations, too, were it not for the fact that this is just S.O.P. (standard operating procedure) for the medical profession, in Honolulu as elsewhere.

Hawaii Society of Internal Medicine

The Hawaii Society of Internal Medicine is a new organization designed to create a representative body for the qualified internists of the Territory. It is a component of the American Society of Internal Medicine, which was founded on April 15, 1956. The fundamental reason for the formation of such a society is the fact that internists are facing economic problems which threaten the future of their specialty. Health insurance plans originated by the private insurance industry, state and Federal Government health plans sponsored by organized medicine, and health insurance laws have all failed to comprehend the role of the internist in American medicine; nor have they provided an economic state in which the internist may continue to practice his specialty.

Recognizing these facts, the internists of Hawaii have been in the process of organizing since October, 1956. There is now a well organized Society of Internal Medicine with some thirty-one members.

The present officers as elected at the last annual meeting are:

President	L. Clagett Beck
President-elect	George H. Mills
Secretary-Treasurer	Shigeru Richard Horio

The prime responsibility of this new society is to insure that all of its members practice good, sound internal medicine, which is based upon a careful history and a thorough physical examination. The second great responsibility is to be alert to all factors—economic, political, and social—which militate against this type of medical practice. This organization is not just another fraternal organization or honorary group.

This society does not consider itself to be in competition with any other medical organization. The internist is under obligation to provide the highest order of professional assistance to other specialty and nonspecialty groups. The internist can be of great aid to the general practitioner in the diagnosis and management of difficult cases. On the other hand it is self-evident that the professional training of the internist could never let him compete with the general practitioner in the fields of obstetrics, minor surgery, or pediatrics, and it is inconceivable that any internist should wish to do so.

Though independent of the American College of Physicians, the American Society of Internal Medicine has been recognized by that body as its economic spokesman. Established committees provide liaison between the two organizations, whose common aim is to secure the finest medical care by means of educational and scientific programs of the American College of Physicians, in a secure economic position made possible by the program of the American Society of Internal Medicine. Both organizations recognize the role of the American Board of Internal Medicine in the development of standards for study, training, and performance.

ELMER C. JOHNSON, M.D.
Past President

This is What's New!

Spirolactone, a steroid, is antagonistic to the salt-retaining steroid, aldosterone. It blocks the ability of aldosterone to retain sodium at the renal tubular level. Spirolactone has caused **diuresis** in all edematous patients who had high urinary aldosterone levels. It was **ineffective** in patients with normal or low aldosterone excretion. (*A.M.A. Arch. of Int. Med.* [Dec.] 1958.)

* * *

The search for a simple test to assist in the diagnosis of **pheochromocytoma** continues. J. T. Wright, of the London Hospital, describes a chemical method of extracting catechol-amines (estimated as noradrenaline). A **catechol acid borate complex** is quantitated **fluorometrically.** In spite of the method being "simple, quick and economical," in the same issue of this journal and working at the same hospital, another investigator described a "simple" biological test for catechol-amines in the urine. (*The Lancet* [Nov. 29] 1958.)

* * *

An increased number of **nonnucleated cells in the urine sediment** (shed from the urethra) indicates **ovarian deficiency** but only in the presence of an intact pituitary and adrenal gland. These epithelial cells without nuclei disappear after hypophysectomy. (*Clinical Endocrinol. and Metab.* [Dec.] 1958.)

* * *

Glomerulonephritis accounts for about **50 per cent** of **adults** with the **nephrotic syndrome.** In the remaining 50 per cent, the nephrotic syndrome is due to any one of some thirty-odd different causes. A meticulous history is the best clue as to the underlying disease. In cases where the history is not helpful, the **renal biopsy** may be very valuable. (*Ann. Int. Med.* [Oct.] 1958.)

* * *

The correction of **ventricular septal defects** has become a rather routine surgical procedure at various vascular centers. The heart and lungs are by-passed by the **heart-lung machine** and **cardiac arrest** induced by **potassium.** The heart-lung machine may take over the patient's cardiopulmonary function for as long as an hour or so, with cardiac arrest for almost the same period of time. The surgical mortality is now a few per cent. The most important additional defect is found to be a **patent ductus,** which makes use of the heart-lung machine impossible until after the ductus is closed, because of the perfused blood backing up through the pulmonary artery. (*British Med. J.* [Dec. 6] 1958.)

* * *

At the international Cancer Congress, Burnet, of Australia, outlined the theory that **cancer** cells are derived from normal cells by **somatic mutation.** He also suggested that **virus** particles produce cancer by altering the genetic make-up of the cell. If the theory is right, then development of cancer is predestined and a single cancer cure is most unlikely. (*Science* [Dec. 12] 1958.)

* * *

A new tetracycline, **demethylchlortetracycline,** which is being referred to as DMCT, "produces much **higher** and **better sustained** levels of antibacterial activity in the serum than tetracycline after single or repeated doses." The co-investigator of the article is Maxwell Finland and the antibiotic was obtained from a mutant of a Streptomyces strain from which chlorotetracycline was originally produced. (*New Eng. J. Med.* [Nov. 20] 1958.)

* * *

Because they lack the ability to agglutinate red cells on routine blood typing, patients with **hypogammaglobulinemia** have been considered "universal recipients." Four such patients with gamma globulin defects were transfused with incompatible red cells. The survival studies revealed **short half-lives** of the transfused cells; none of them could be considered "universal recipients." (*Blood* [Jan.] 1959.)

FRED I. GILBERT, JR., M.D.

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In Memoriam -- Doctors of Hawaii -- XIX

This is the nineteenth installment of In Memoriam—Doctors of Hawaii.

Alexander Montague Atherton

Alexander Montague Atherton was born in Honolulu on August 13, 1875, the son of Mr. and Mrs. Joseph B. Atherton.

He attended Punahou Preparatory School and Oahu College, graduating in 1893. In September of the same year he entered Wesleyan University, Middletown, Connecticut, and received his A.B. in June, 1897. From there he went to Johns Hopkins Medical College which granted him his M.D. in 1901.

In a competitive examination for appointment to New York City Hospital, Dr. Atherton was one of eight out of the thirty contestants to receive an appointment.

While visiting in the Islands during the summer of 1901, Dr. Atherton passed his Medical Board examinations and received his license to practice in the Territory. During September and October of the same year he went to Hamakua to take over the practice of Dr. Greenfield, who had broken his leg.

Dr. Atherton returned to New York to begin his internship in December, 1901. During the winter he contracted a heavy cold which settled in his lungs. He kept on with his work, however, until June, 1902, when a succession of hemorrhages forced him to go to a sanitarium in the Adirondacks.

In April, 1903, Dr. Atherton returned to Honolulu to see his father, arriving the morning following the latter's death. He remained three months but the climate proved unfavorable for him and he returned to New York and entered Dr. Loomis' sanitarium at Liberty. There, in spite of all efforts, he continued to fail.

On August 18, 1903, he married Miss Ellen Louise Baker of Providence, Rhode Island.

Ten days later on August 28, 1903, Dr. Atherton died at Liberty, New York, at the age of 28.

William Lineas Maples

William Lineas Maples was born at Sevierville, Tennessee, on March 31, 1869. He was the son of Edward and Martha Jane (Runions) Maples.

He was a member of the first class to graduate from the city school in Knoxville, Tennessee, June 8, 1888. For excellence in oratory he was awarded the Dodson Medal. From 1888-1889 he taught school in Austin.

Following this he entered the medical department of Howard University in Washington, D. C. At the same time he received an appointment as a clerk typist in the federal pension office.

After his graduation from Howard in 1892, Dr. Maples returned to Knoxville where he practiced until 1901.

During that period Mayor S. C. Heiskell of Knoxville appointed Dr. Maples commissioner of Negro exhibits at the Tennessee centennial.

At the time of the Spanish-American War, Dr. Maples served as hospital steward with the Hospital Corps of the Third Regiment of North Carolina Volunteers.

Coming to Hawaii in 1901, Dr. Maples was employed in the hospital of the Hawaiian Commercial and Sugar Co. of Puunene, Maui, until 1931.

Dr. Maples died at Wailuku on January 22, 1943, at the age of 73.

He was past Chief Ranger of the Court of the Foresters of the Valley Isle.

Charles Raymond McLean

Charles Raymond McLean was born at Princeton, Illinois, on September 14, 1877.

He was a graduate of Princeton High School and received his medical training at Illinois University, Urbana, and at Rush Medical College from which he graduated in 1901.

After graduation Dr. McLean came to the Islands and was a plantation doctor on Hawaii and at Koloa, Kauai, for about three years. Sometime between 1904 and 1905 he moved to Oahu where he became physician for the Ewa Plantation Company.

In 1906 in Honolulu Dr. McLean married Anne Elizabeth Potts of Wilkes-Barre, Pennsylvania. The couple were the parents of one son, George Stephen.

The doctor served at Ewa Plantation until 1928 when he became a government physician at Waianae for two years until his retirement in 1930.

Dr. McLean died August 7, 1942, in Honolulu, within little more than a month of his 65th birthday.

He was a member of the Hawaii Territorial Medical Association, serving as President in 1925, and was a Mason, Shriner, and Knight Templar.

Jiro Yoshizawa

Jiro Yoshizawa was born in Tochigi prefecture, Japan, on November 5, 1879, and in 1900 he received his medical degree from Tokyo Imperial University.

Arriving in Honolulu in September, 1903, Dr. Yoshizawa was licensed to practice in the following year. By 1907 he was practicing at Kealakekua, Hawaii, and in 1909 moved to Koloa, Kauai, where he was in practice for twelve years.

Dr. Yoshizawa returned to Japan in 1921 where he spent a year in postgraduate studies. On his return he settled in Honolulu until 1937 when he moved to Paia, Maui. During World War II, he was interned on the Mainland as an enemy alien. On being released in 1945, he returned to Honolulu but retired from active practice.

The doctor was married and the father of three sons: Hironori, Rintaro, and Susumu and four daughters: Mrs. Toshi Ono, Mrs. Toku Umehara, Mrs. Tomo Ramos, and Mrs. Isayo Furukawa.

Dr. Yoshizawa died on September 26, 1957, in Honolulu at the age of 77.

During his lifetime he was active in community affairs, having been President of the Kona Hongwanji Gakuen while on Hawaii and of the Kauai Shinpo Sha in Koloa. In Honolulu he was President of the Board of Directors of the McCully Japanese School when it was founded. He also served as an officer of the Japanese Medical Association and was a member of the Honolulu County Medical Society.

The Honolulu County Medical Library

President's Report

Members of the Board during the year were: Drs. Richard Ando, Grover Batten, Harold Civin, Paul Gebauer, Henry Gotshalk, Clifford Kobayashi, John Lowrey, Chew Mung Lum, Thomas Min, Harold Moffat, Andrew Morgan, and Dean Walker.

During 1958, 275 new books were added, and 654 journals are currently being received (over 600 of these are monthlies). The total number of volumes in the collection now numbers 25,081, an increase of 2,006 volumes over 1957.

This growth, together with the return to our quarters of those journals previously stored in the office of the Hawaii Medical Association, caused such cramping that it was necessary for additional journals to be put in dead storage. Dr. Hastings Walker kindly gave us space in the basement of Leahi Hospital where they must remain for the duration.

Agreement was reached with The Queen's Hospital about the site of the new building. The new library will be next door to the Mabel Smyth Building where Hale Kula now stands. The Nursing School is going to be housed in new quarters above the new outpatient department now under construction at Queen's.

Mr. Vladimir Ossipoff has guaranteed plans and specifications that will be within the $300,000 budget. The first set of preliminary plans is due momentarily.

The Library Endowment Fund is worth about $148,000. In time the income from it could go toward the annual support of the new building.

GROVER H. BATTEN, M.D.

Report of Fund-raising Committee

Numerous mainland foundations were approached in an attempt to raise funds. This attempt was very disappointing. The only funds obtained were from Pfizer and Company.

The next attack was on the local doctors, and, at the present time, a total of 196 doctors have contributed a total of $21,172. This leaves a total of 268 doctors from whom we still hope to obtain contributions.

Our next appeal was to the local foundations and we acknowledge with gratitude their generous support. All the local foundations contacted have donated generously, to a total of $26,750.

In December, 1958, 2,320 letters were sent out to individuals and a total of 5,543 letters were sent out to local business houses. The responses to this appeal are still coming in. A total of $1,165 has been received from individuals and $1,047 has been received from local corporations.

At the present time a total of $43,403.90 has been received and if pledges are included, the total is raised to $50,134.90.

The Honolulu County Medical Society has agreed to turn over $30,000 to the Library Fund from "Operation Hypo" and so if this is included, at the moment we now have received or had pledged a total of $80,134.90.

It is extremely important that the support among doctors be increased and that all doctors take up this project among the community. It is impossible for your Fund-Raising Committee, of only three members, to contact everyone and the active participation of the entire membership of the Library Corporation is earnestly solicited.

JOHN J. LOWREY, M.D.

MINUTES OF ANNUAL MEETING

January 13, 1959, at 7:30 P.M.
Mabel Smyth Lounge

Dr. Batten presided, and there were approximately 25 members present (15 members constitute a quorum).

PRESIDENT'S REPORT Dr. Batten reported on the progress of the Library during the past year.

AUDITOR'S REPORT Mr. Frank Leman, our auditor, made a report on the financial status of Library funds. He remarked that his quotations were approximate, since the fiscal year ends February 28, 1959. The market value of the Endowment Fund has increased from $127,000 to $148,000. Expenses in our General Fund appeared to be well within our income. The Building Fund receipts are in a small checking account and in several time deposits at the Bank of Hawaii.

FUND-RAISING COMMITTEE REPORT Dr. Lowrey briefly reviewed the work his committee had done in raising funds for the new building. Dr. Henry inquired about the billing on pledges and asked about further efforts of Team Captains. Dr. West suggested a long-term pledge payment plan. Dr. Lowrey said the Board had under consideration a new approach to all noncontributors, and asked for the kokua of all present.

BUILDING COMMITTEE REPORT Dr. Civin stated that his committee was composed of himself as chairman, Drs. Morgan, Walker, and Dr. Batten, *ex officio*. He reported that the Building Committee had met frequently

(Continued on page 428)

Book Reviews

The Chemical Prevention of Cardiac Necroses

By Hans Selye, M.D., Ph.D., D.Sc., 235 pp., $7.50, The Ronald Press Company, November, 1958.

This is a very interesting monograph. One of the most brilliant endocrine investigators of our time documents carefully over 30,000 experiments which were carried out as a result of some observations that if a rat is prepared with what is ordinarily an innocuous amount of a cortico-steroid, and then given certain electrolytes (especially those containing sodium), the animal will develop focal myocardial necroses.

This interesting phenomenon was studied in many different ways. Many different electrolytes were used, mostly containing sodium—the character of the resulting lesion depends largely upon the anion.

Further observations revealed that $MgCl_2$ and KCl can protect the myocardium against the necrotizing effects of a great many agents. The work is presented in an orderly fashion, and despite the apparently bewildering amount of data listed, the monograph is quite readable.

The author is careful to warn that the work as yet has little clinical application, but the close correlation between the lesions of a great many different cardiac maladies (Fiedler's myocarditis, chronic primary myocarditis, uremic cardiopathy, etc.) and those produced by sensitization by cortico-steroids followed by sodium-containing eletrolytes, deserves careful attention and the ultimate use of magnesium and potassium in clinical medicine (for their protective action) may not be too far away.

A. S. HARTWELL, M.D.

So You Have Glaucoma

By Everett R. Veirs, M.D., 64 pp., $2.75, Grune & Stratton, 1958.

This is a down-to-earth, easily understood, concise book which explains the types, causes, symptoms, diagnosis, and treatment of glaucoma. It will undoubtedly save the busy ophthalmologist a great deal of time by providing the correct answers to the many questions of his worried patients. The Do's and especially the Don'ts in the postscript apply not only to most patients with glaucoma but also to the harrassed, hurried, nervous multitude of Twentieth Century men in need of tranquilizers.

WILLIAM JOHN HOLMES, M.D.

The Eternal Search

By Richard R. Mathison, 381 pp., $5.95, G. P. Putnam's Sons, 1958.

The Eternal Search is a Paul De Kruif treatment of the history of medicine, written by a newspaper reporter whose appreciation of the bizarre far exceeds his penchant for accuracy.

In twenty-four chapters and sixteen pages of illustrations, the oddities of medical arts throughout the ages are catalogued. Comparisons are made with the thought and practices of the present—and this feature, rather than adding interest, sorely detracts from the text, since the comments vary from trite to banal, attempting a touch of humor.

In spite of these detractions, and because the history of medicine has inherent values that can surmount even the newspaper treatment, the book is worth cursory reading. Some interesting bits of lore are to be found, of the sort that makes interesting conversation while closing in the OR. The author must be commended for his chapters on burial practices and treatment of the mentally sick.

The book cannot in any sense be considered a source of historical research material. For older children, or anyone not acquainted with medical history, it would provide a palatable outline of the subject, in the best *Reader's Digest* tradition.

FRANK C. TABRAH, M.D.

★ Spinal Cord Compression

By I. M. Tarlov, M.D., 127 pp., $7.50, Charles C. Thomas, 1957.

This monograph summarizes the ingenious and beautifully carried out experimental work which Dr. Tarlov has done on the results of acute and gradual compression of the spinal cord. A good deal of this material has been published previously in various journals. He gives an experimental explanation for the known clinical syndromes so frequently seen of poor recovery following acute compression and excellent recovery following gradual compression of the spinal cord if operative relief can be accomplished. The work shows the microscopic lesions that are produced and also gives a rationale for clinical treatment on the basis of the experimental work.

This is an excellent monograph and highly recommended for anyone interested in the field.

JOHN J. LOWREY, M.D.

Noise and Your Ear

By Aram Glorig, Jr., M.D., 146 pp., $6.50, Grune & Stratton, 1958.

An excellent book on acoustics. The measurement of hearing is discussed in detail. Noise is explained in relation both to industry and to hearing loss. It is recommended reading for the otologist.

BARTON R. BECKER, M.D.

★ Tumors and Tumorous Conditions of the Bones and Joints

By Henry L. Jaffe, M.D., 629 pp., $18.50, Lea & Febiger, 1958.

Those who have looked forward with anticipation to the publication of this book will be gratified to know that their enthusiasm was justified. The author has based the work upon a long and fruitful experience at the Hospital of Joint Diseases. Although much of the material has already appeared in the form of separate papers, this is not a product of the paste pot. Both common and uncommon conditions are described from the clinical, radiological, and pathological points of view. The text is illustrated by many photographs of high

(Continued on page 438)

★ means "highly recommended."

Hawaii Medical Association

A.M.A. Delegate's Report

I attended both sessions of the twelfth Clinical Meeting of the A.M.A., in Minneapolis, December 2 to 4, 1958.

Commission on Medical Care Plans. The report of this four-year-old Commission was the most important item discussed; action on it was deferred, however, on the ground that it had (because of its cost) been distributed only to delegates and not to constituent associations and it had therefore not been widely read. It is to be acted upon in June. We are requested to act on it by April 1. We are asked by the House of Delegates to answer these questions:

1. Free Choice of Physician—Acknowledging the importance of free choice of physician, is this concept to be considered a fundamental principle, incontrovertible, unalterable, and essential to good medical care without qualification?

2. Closed Panel Systems—What is or will be your attitude regarding physician participation in those systems of medical care which restrict free choice of physician? These suggestions "question," the House of Delegates said, "whether attitudes toward free choice of physician and the closed panel system may be undergoing evolutionary change."

Evidence that these attitudes *have* been undergoing *some* changes abounds in Part I of the Commission's report—which runs to 117 pages. Part II contains the statistical appendices. Thirteen physicians, most of them distinguished in the A.M.A. hierarchy, and two laymen (Jay Ketchum and E. J. Faulkner) comprised the Commission under the chairmanship of Leonard Larson of North Dakota, Chairman of the Board of Trustees. They made a detailed, painstaking study of closed panel plans in New York, St. Louis, Chicago, Washington, D. C., Oakland, San Francisco, Palo Alto, Los Angeles, and Beckley (W. Va.). Other types of plans were also studied in similar detail, and comparisons were made. The report is so objective, and takes such care to avoid sweeping conclusions, that it is impossible to summarize; but it appears to express the view, on the whole, that closed panel plans are not necessarily an inferior way to practice medicine. I counted 21 compliments for them in the text of the report.

A.M.A. Objectives and Basic Programs. The complex and bulky report of the committee appointed last year to study this immense subject was received and commended. One of the committee's recommendations—to amend Article II of the Constitution (as follows)—was approved by the House: "The objectives of the Association are to promote the science and art of medicine and the betterment of public health and an understanding of the socio-economic conditions which will facilitate the attainment of these objectives."

Health Care of the Aged. Called for by the House of Delegates is the development of an effective voluntary health insurance or reduced-income prepayment program "for persons over 65 years of age with reduced income or very modest resources" with a reduced premium rate made possible by physicians' agreement to accept lower fees for this group. Cooperation of Blue Shield and private insurance programs is being requested.

Osteopathy. An attempt to assert states' rights in the matter of ethical strictures against osteopaths was rejected, but re-study of the problem with a report in June was ordered.

United Funds. United Fund organizations, slapped down last June, were raised to their feet and dusted off by a statement that the A.M.A. neither approves nor disapproves of them. High-level study of the argument was ordered.

Medical Education. The A.M.A. officially recognized the need for more medical schools and more doctors.

Administrative. The Chicago Office now has seven divisions: Business, Law, Communications, Field, Scientific Publications, Socio-Economic Activities, and Scientific Activities. A new Council (permanent committee) on Legislative Activities will take over operation of the Washington Office, operating from Chicago.

A variety of miscellaneous actions are summarized in the *A.M.A. News* of December 15 and will be reported in detail in the *J.A.M.A.*

HARRY L. ARNOLD, JR., M.D.
A.M.A. Delegate

MINUTES OF THE COUNCIL MEETING

January 16, 1959, at 6:30 P.M.
Naniloa Hotel, Hilo

PRESENT:

Dr. William N. Bergin, presiding; and Drs. T. Nishigaya, E. F. Cushnie, R. C. Yap, C. M. Burgess, Frank Spencer, Kenneth Fujii, S. Mizuire, and J. A. Burden, plus members of the Hawaii County Medical Society and guests, Dr. Thomas H. Richert and Mr. R. M. Kennedy.

ARRANGEMENTS COMMITTEE REPORT:

The Chairman of the Arrangements Committee, Dr. Henry Yuen, reported that Dr. Ota and the Woman's

Auxiliary were in charge of hospitality; Dr. Helms, transportation; Dr. Pete Okumoto, American Legion Hall, where the meetings are to be held; Dr. Miyamoto, hotel reservations; and Dr. Oto, golf. He said that the Hawaii County members were planning an outstanding social program and would, with the help of the Woman's Auxiliary, provide transportation for all the visitors.

GROUP LIFE INSURANCE:

Mr. Richard Kennedy, Executive Secretary of the Honolulu County Medical Society, reported that Honolulu is making a survey to see what type of group life insurance plan would be acceptable to the doctors; that is, whether they want ten, fifteen, or twenty thousand dollar policies, and whether they want the premium to remain constant throughout the life of the policy or whether it should be increased with age or the amount of insurance decrease with age.

LEGISLATIVE COMMITTEE:

Dr. Yap said he felt that this committee should be on a Territorial level because its actions affect every doctor in the Territory. That it has been the policy of the Committee to take from the hopper all the legislation which involves the welfare of physicians in the Territory. The Committee does not let its own sentiments enter into their decisions. He suggested that every county president in office when the legislature is in session become a member of the Committee. Often the committee chairman has to contact every member and the president is the logical one to report the opinions of his county society members. With reference to funds, he said it was moved at the last House of Delegates meeting to set aside $1,000 for the use of this committee. It was Dr. Yap's opinion that these funds should be used only for the Legislative Committee for entertainment, communications and runners. Dr. Yap concurred with the report that the Chairman should serve four years.

ACTION:

Dr. Burden moved, seconded by Dr. Spencer, that the Chairman be appointed by the President of the Hawaii Medical Association and that a member from each island, the president of each county society, and the President of the Board of Health be made members of the Committee. The motion carried.

Dr. Mizuire asked if the Committee was also aggressive and Dr. Yap advised that any island could institute a bill and if it is of benefit to every doctor in the Territory, the Committee supports it.

Dr. Richert explained that he had been waiting to make appointments in Honolulu pending action of the Council. He agreed that the Legislative Committee should be a territory-wide committee and said he thought the members who had already accepted would be satisfactory.

The matter of who should do the appointing was next discussed and it was the feeling of the Council that since it is an HMA committee, the president of the HMA should make the appointments to it.

ACTION:

Dr. Spencer moved, seconded by Dr. Burgess, that the Committee elect its own chairman. The motion carried.

Dr. Spencer moved, seconded by Dr. Burgess, that we not appoint any subcommittee at this time and that this matter should be taken up at the next annual meeting. The motion carried.

Dr. Cushnie moved, seconded by Dr. Nishigaya, that the Legislative Committee have the authority to utilize the money at its discretion. The motion carried.

CHRONIC ILLNESS COMMITTEE:

Dr. Bergin reported on the activities of the Chronic Illness Committee. Mr. Veltmann was asked to talk on the subject of prepaid medical care for people over 65. He said that Honolulu County had asked him to prepare a plan and present it to them for review. He said that Blue Shield is proposing a schedule that should be used nationally and that he proposes to present this to Honolulu's committee as soon as it is received. He said he understood that this was going to be extended to commercial plans and didn't understand why and asked Mr. Kennedy to explain further.

Mr. Kennedy said the Honolulu Medical Care Plans Committee did not feel they were in a position to draft such a plan and so they asked the commercial plans if they were interested and also the HMSA the same thing. Letters are now out on this but replies have not been received.

ACTION:

Dr. Nishigaya moved, seconded by Dr. Cushnie, that the Council go on record as approving of the principle of the plan and await the results of the investigation of Honolulu County before acting on it further. The motion carried.

HAWAIIAN SCIENCE FAIR:

Dr. Bergin advised that the HMA donated $50.00 to the last science fair and that the House of Delegates recommended that this year we contribute $100.00 to the Fair and, in addition, offer prizes for an exhibit or exhibits in the field of biology or medicine.

ACTION:

Dr. Burgess moved, seconded by Dr. Nishigaya, that we give a $50.00 prize to be distributed at the discretion of the officers of the HMA. The motion carried.

ROSTER:

Dr. Bergin discussed the matter of the proposed roster and outlined the anticipated cost.

ACTION:

Dr. Spencer moved, seconded by Dr. Mizuire, that the matter of the roster be referred back to the House of Delegates for reappraisal. The motion carried.

AAPS ESSAY CONTEST:

The AAPS Essay Contest was next discussed. Mr. Kennedy said that had they known it was going to be on a territorial level, they would have kept their amounts down.

ACTION:

Dr. Burgess moved, seconded by Dr. Nishigaya, that the Council authorize the Woman's Auxiliary to offer one $100.00 prize. The motion carried.

REIMBURSEMENT OF COUNCILORS' EXPENSES:

Dr. Bergin explained that in the past the councilors' expenses had been reimbursed to the different county treasuries.

ACTION:

Dr. Nishigaya moved, seconded by Dr. Cushnie, that the Councilors be reimbursed direct rather than through the counties. The motion carried.

(Continued on page 414)

Notes and News

This column is written for your information and entertainment about your professional, scientific, and social accomplishments. If you have any newsworthy items, please telephone the News Editor, Dr. Masato M. Hasegawa, or his secretary, at 58-296, or mail them to 1010 So. King Street.

Doctors and Our Community

Dr. Edwin Kam, Windward Oahu Orchid Society President, spoke at the bi-monthly workshop meeting of the Honolulu Orchid Society in December.

Dr. Richard W. You, Honolulu physician who served as a doctor on the United States 1956 Olympic team, spoke on January 22, 1959, to the Honolulu Optimist Club about the Women's Track and Field events in the 1960 Olympics.

Lt. Col. Carl W. Hughes, Tripler Army Hospital surgeon, was presented the nation's top award in the field of military medicine, The Sir Henry Wellcome Award, at the annual meeting of the Association of Military Surgeons held in Washington, D. C. The Wellcome award was won three years ago by another Tripler doctor, **Col. Warner F. Bowers.**

Dr. David L. Pang was elected President of the Chinese Chamber of Commerce of Hawaii on December 3, 1958. He is a general practitioner at the Nuuanu Clinic.

Dr. David L. Pang was succeeded by Theodore C. H. Char, as president of the Birthday (Pun Tao) Club recently. **Dr. H. Q. Pang** was elected vice-president. **Dr. Thomas Y. K. Chang** is the co-chairman of the membership committee.

Dr. Richard W. You attended the National A.A.U. and U. S. Olympic convention as Hawaii's delegate in Chicago on December 4 to 7, 1958.

Dr. Robert B. Faus was named the new Potentate of Aloha Temple AAONMS, leader of Nobles of the Mystic Shrine in Hawaii, Guam, and Okinawa.

Col. Warner F. Bowers and **Lt. Col. Carl W. Hughes,** Chiefs of Surgery at Tripler Army Hospital, are writing books on surgery. Col. Bowers is working on "Surgical Gastroenterology." Col. Hughes is working on "Surgical Philosophy in Mass Casualty Management."

Our New Chiefs

Dr. Robert Stafford Spencer has been appointed Medical Director of the Territorial Hospital at Kaneohe effective January 1, 1959. Dr. Spencer has been Acting Director since the resignation last August of **Dr. Robert A. Kimmich,** who is now Clinical Director at a Chicago research institute.

Brigadier General Richard L. Bohannon arrived at Hickam Air Force Base to take over new duties as Command Surgeon for the Pacific Air Forces. He replaces **Major General Oliver K. Niess,** who left Hickam in November for Washington, D. C., for duty as U. S. Air Force Surgeon General.

Dr. Paul Gyorgy arrived during the latter part of December to serve as Visiting Professor of Pediatrics at Children's Hospital. He is Professor of Pediatrics at the University of Pennsylvania School of Medicine and Chief of Pediatrics at Philadelphia General Hospital.

Dr. Nathan Shklov has joined Waialua Agricultural Co. as Assistant Medical Director.

Dr. L. Q. Pang has been elected President of the medical staff of St. Francis Hospital. **Dr. Varian Sloan** is the Vice-President, **Dr. Richard D. Moore,** Secretary, and **Dr. Bernard Yim,** board member. **Dr. Edward Lau** was named Chief of Surgery, and **Dr. Philip Watt,** Chief of Pediatrics.

Dr. Thomas H. Richert was installed as the 1959 President of the Honolulu County Medical Society on December 2, 1958. **Dr. H. Q. Pang** was installed as President-elect, **Dr. A. S. Hartwell,** Secretary, and **Dr. O. D. Pinkerton** as Treasurer.

Dr. Paul W. Gebauer was elected President of the Hawaii Chapter, American College of Surgeons, at a recent annual meeting in the Princess Kaiulani Hotel. Other officers: **Dr. Rogers Lee Hill,** Vice-President, **Dr. F. J. Pinkerton,** Secretary-Treasurer, and counselors, **Dr. John J. Lowrey, Dr. Shoyei Yamauchi,** and **Dr. Verne C. Waite.** Four new Fellows of the College were honored: **Dr. Scott C. Brainard, Dr. William E. Iaconetti, Dr. Merton H. Mack,** and **Dr. Frederick B. Warshauer.**

Dr. Lester T. Kashiwa of Wailuku has been elected President of the Maui County Medical Society. **Dr. Seiya Ohata** of Paia was elected Vice-President, and **Dr. Ah Yet Wong** of Wailuku was elected Secretary-Treasurer.

The Territorial Association of Plantation Physicians elected **Dr. Edward B. Underwood,** physician for Hawaiian Commercial and Sugar Company, as President. Vice-President is **Dr. Richard Y. Noda** of Oahu Sugar Company, and Secretary-Treasurer, **Dr. Garton E. Wall** of Ewa Plantation Company.

Public Service

On November 28, 1958, **Dr. Carl E. Johnsen, Jr.** introduced on TV a film demonstrating the technique of mouth to mouth resuscitation. This was presented by the Hawaii Medical Association.

Dr. Robert G. Benson is the local fund-raising chairman in a national drive for the University of Wisconsin Medical School Library.

Dr. Harold Civin, head of the pathology laboratory at The Queen's Hospital, spoke on "What You May Get for Your One Billion Dollars of Medical Research" at the Sigma Xi sponsored University of Hawaii series of "Hawaii's Place and Future in Scientific Research" on November 20, 1958.

Dr. L. G. Stuhler directed the 1958 community-wide Diabetes Detection Drive, which was held during the Diabetes Week, November 16 to 22.

Dr. Varian Sloan, past chairman of the Honolulu County Medical Society Public Service Committee, estimated that a total of 115,702 Salk anti-polio shots were administered in the total Operation Hypo program.

Dr. Nils P. Larsen of the Hawaiian Academy of Science was presented by Hiram Fong, President of the Grand Pacific Life Insurance Co., with $1,000 for two company scholarships to the Academy. The scholarships will be awarded to winners of the senior division, second annual Hawaiian Science Fair. Another $1,000 in savings bonds will be given to other high school winners.

Dr. Morton E. Berk spoke on "Effects of Physical Fitness" at the Central YMCA recently. In the same series of lectures, **Dr. James G. Harrison** talked on "The Mental Attitude in Physical Fitness."

103rd ANNUAL MEETING
HAWAII MEDICAL ASSOCIATION

APRIL 23-26, 1959

HILO, HAWAII

FIRST ANNUAL MEETING

TO BE HELD SINCE THE ADMISSION

OF THE 50th STATE

STATE of HAWAII

HAWAII MEDICAL ASSOCIATION
103rd ANNUAL MEETING
HILO, HAWAII • APRIL 23 TO 26, 1959

Registration at the American Legion Hall—April 23—1:00- 2:00 P.M.
April 24—7:30-11:00 A.M.
April 25—7:30-11:00 A.M.

Registration Fee—$10.00

THURSDAY APRIL 23	2:00 P.M.	House of Delegates and General Membership Meeting—American Legion Hall
	6:00 P.M.	Cocktails and Buffet Supper—Hilo Yacht Club—Hawaii County Medical Society hosts
FRIDAY APRIL 24	7:30 A.M.	Breakfast ($2.00) American Legion Hall
	8:30 A.M.	Panel on *Coping with the Teenager* MODERATOR: Dr. J. Roswell Gallagher (guest speaker from Boston, Mass.) PANELISTS: Drs. Halbert L. Dunn (guest speaker from Washington, D. C.), Donald C. Marshall, William Mayer, Kenneth H. Rusch
	9:10 A.M.	*Rehabilitation of the Hemiplegic* by Dr. R. Frederick Shephard
	9:20 A.M.	*Cerebral Hematoma* by Dr. John J. Lowrey
	9:30 A.M.	Intermission to view exhibits
	10:00 A.M.	Panel on *Hernia Hydrocele and Undescended Testicle* MODERATOR: Dr. Tague C. Chisolm (guest speaker from Minneapolis, Minn.) PANELISTS: Drs. Charles S. Judd, Jr., Edward K. Lau, James A. Mitchel, Shizuto Mizuire
	10:45 A.M.	*Portal Hypertension, Experiences in the Management of Hemorrhage from Esophageal Varices* by Dr. Gilbert C. Freeman
	10:55 A.M.	*Improving Medical Certifications of Death* by Dr. Halbert L. Dunn
	2:00 P.M.	House of Delegates—American Legion Hall
	7:00 P.M.	Presidential Address by Dr. William N. Bergin
	7:30 P.M.	*Problems in Steroid Therapy* by Dr. Philip S. Hench (guest speaker from Rochester, Minnesota)
	9:30 P.M.	Introduction of new officers
SATURDAY APRIL 25	7:30 A.M.	Breakfast ($2.00) American Legion Hall
	8:30 A.M.	Arthritis Clinic conducted by Dr. Philip S. Hench (sponsored by Merck, Sharp & Dohme)
	9:30 A.M.	Intermission to view exhibits
	10:00 A.M.	*Pitfalls in Surgery of the Hand* by Dr. Leabert R. Fernandez
	10:10 A.M.	*Surgical Approach to Fascial Space Infection in the Hand* by Dr. Raymond G. Chang
	10:20 A.M.	*Study of Lipoproteins in the Nisei* by Dr. Kyuro Okazaki
	10:30 A.M.	*Report on Coronary Atherosclerosis in our Different Ethnic Groups from a Successive Series of 2,000 Autopsies* by Dr. Nils P. Larsen and Dr. Walter M. Bortz
	12:00 M.	Luncheon Meeting—Hawaii Territorial Academy of General Practice at the Hukilau Hotel
	6:00 P.M.	Cocktails and Dinner Dance $7.50 at the Volcano House. Dinner Jackets optional. Entertainment courtesy of the Hawaii County Medical Society
SUNDAY APRIL 26	7:00 A.M.	Breakfast at Hilo Country Club
	7:30 A.M.	Annual golf tournament, Hilo Country Club
	12:00 M.	Picnic at Hilo Country Club ($2.50)

Gratuities are included in the price of all tickets

WOMAN'S AUXILIARY ACTIVITIES

FRIDAY APRIL 24	9:30 A.M.	Registration, Naniloa Hotel
	10:00 A.M.	Auxiliary Executive Board Meeting
	12:30 P.M.	Luncheon honoring Mrs. William R. Underwood, National President, Naniloa Hotel
	2:00 P.M.	Tour to Puna Lava Flow (2 hours)
	7:30 P.M.	General Auxiliary Meeting, Naniloa Hotel
SATURDAY APRIL 25	8:30 A.M.	Woman's Golf Tournament

Dr. William A. Myers, pediatrician with the Straub Clinic, was honored on the evening of November 22, 1958, when the Order of DeMolay conferred upon him its highest award, the Legion of Honor.

The *Honolulu Star-Bulletin* featured a picture on November 11, 1958, in which were the guests, **Dr. and Mrs. Andrew C. Ivy, Jr.** and **Dr. and Mrs. Douglas Murray.** The occasion was the opening of the Church of the Holy Nativity's new Youth Center.

The *Honolulu Star-Bulletin,* on December 13, 1958, also featured a story and picture of the old Straub Clinic. The picture showed **Dr. George Straub** pointing out his old home and office. The building, occupied by the Child and Family Service until recently, has been completely demolished for a parking area.

Medical Progress

A construction boom of medical facilities in the Territory is on. The Queen's Hospital is in the midst of constructing a $3 million portion of an ultimate $4½ million expansion.

Territorial Health Department is to have a $1 million new building, probably in 1960.

Hilo Memorial Hospital is to have a $1 million new wing plus tuberculosis bed conversion.

Maunalani Hospital and Convalescent Center is to have a $900,000 new wing.

Kuakini Hospital is to have a $750,000 new wing.

St. Francis Hospital is having a new $500,000 wing.

The Blind Rehabilitation Center will have a $250,000 new building with workshop.

The heart-lung machines at St. Francis and The Queen's Hospitals were featured in the *Star-Bulletin* on January 6 and in the *Advertiser* on February 13, 1959. **Dr. Fred I. Gilbert, Jr.** and **Dr. Sumner Price** of The Queen's Hospital were demonstrating the latter.

A project, the five-year study of tuberculosis cases, dropped from the Department of Health register after five years of inactivity, was started under the direction of **Dr. Robert H. Marks** by the Oahu Tuberculosis and Health Association.

Doctors as Athletes

Dr. Ted Tomita carded 78-11—67 to annex the afternoon sweepstakes of the Waialae Country Club recently.

Dr. M. E. Stevens was tied for first place in a golf match play against par of the Oahu Country Club. He was even with par.

Hobbies, Wives, and Everything

The Woman's Auxiliary to the Honolulu County Medical Society will process entries for the essay contest sponsored by the Association of American Physicians and Surgeons' Freedom Programs. This is the first time that high school students in Hawaii have been eligible to participate in the national contest. The essays may be written on either of these topics: "The Advantages of Private Medical Care," or "The Advantages of the American Free Enterprise System." **Mrs. Robert Dimler** is in charge of the contest for Oahu. Assisting Mrs. Dimler are the **Mesdames Charles Judd, Donald Brown, Carl Youngkin, Joseph Lucas, Eugene McGinnis, Elmer Johnston** and **Merton Mack.**

Twenty-three new members were honored at the annual meeting of the Woman's Auxiliary to the Honolulu County Medical Society. New officers elected were: **Mrs. George B. Garis,** President; **Mrs. Herbert G. Pang,** President-elect; **Mrs. Joseph T. Lucas,** Vice-President; **Mrs. Shigeru Horio,** Recording Secretary; **Mrs. William H. Stevens,** Corresponding Secretary; **Mrs. Harold T. Kimata,** Treasurer; **Mrs. Gilbert C. Freeman** and **Mrs. Kyuro Okazaki,** Members at Large. The meeting started with a tour of the home and garden of **Dr. and Mrs. Niall M. Scully,** a beautiful creation in Waialae-Kahala, then the group proceeded to the lovely home of **Dr. and Mrs. Louis L. Buzaid,** at Kaikoo Place, to view the Japanese rock garden and Oriental court where refreshments were served.

Mrs. Robert Johnston's Ch. Amandre of Holligrove won the best of variety in miniature poodles during the recent Poodle Club of Hawaii showing. Another outstanding performer was **Dr. Richard D. Moore's** Sunnyheath La Petite.

Change of Scene

Dr. Harold T. Kimata announced the resumption of practice in November, 1958.

Dr. Henry Yokoyama announced the removal of his office to 1507 S. King St.

Dr. Stanley E. Kobashigawa announced the opening of his office in Pearl City.

Dr. Edward C. K. Lum announced the opening of his office at 181 S. Kukui St.

Dr. Tell Nelson announced his return to active practice in November, 1958.

Dr. Donald Char resigned as Director of Medical Education at Children's Hospital to join the staff of the University of Washington Medical School.

Occupational Health

The first International Congress on Occupational Health to be held in the Western Hemisphere will take place in New York City, July 25 to 26, 1960. Dr. Leo Wade of New York is Chairman of the Organizing Committee.

The Scientific Program Committee invites submission of papers for presentation at the Congress on the following aspects of occupational health: administrative practices, medical practices, surgical practices, education and training, social and legal aspects, environmental hygiene, influence of environmental factors in health, work physiology and psychology, specific industries, and general aspects. Those desiring to present papers must submit abstracts before January 1, 1960.

Wellness

After participating in the annual meeting of the Hawaii Medical Association, Dr. Halbert L. Dunn, chief of the National Office of Vital Statistics, will deliver a series of lectures from April 27 to May 1 on the subject of positive health—i.e., levels of "wellness." Generally, we speak about health in negative terms such as morbidity and mortality. Dr. Dunn says that we should pay more attention to "wellness" and learn to measure it.

Pan-Pacific Surgical Association Eighth Congress Honolulu, Hawaii, Sept. 28-Oct. 5, 1960

The Eighth Congress of the Pan-Pacific Surgical Association will be held in Honolulu, Hawaii, September 28 through October 5 in 1960.

(Continued on page 418)

County Society Reports

Kauai

The regular monthly meeting of the Kauai County Medical Society was held on the 2d of December, 1958, Tuesday, 7:38 P.M. at the Lihue plantation Training Center. Visitors present were: Drs. Maag, Zandee, and Quisenberry.

Dr. Worth exhibited a booklet for diabetics with menus for the different racial groups that would be available to any physician interested. He also spoke on the review of the tuberculosis situation as it now exists on the island of Kauai. On Kauai in 1955-57 the incidence of tuberculosis was 2.4 per thousand, or about 2½ times the Territorial incidence. Dr. Worth attributed this increased number of new cases to (1) a very thorough follow-up and tenacious investigation conducted by Dr. Kim, (2) a real difference in incidence on this island. He also said that 68 per cent of all cases are found by survey films, and that the age incidence is nearly all in the 25 years-and-up group. For this reason he suggests that we discontinue the chest x-ray of high school graduates and that we tuberculin test children in the grade school instead. Dr. Cockett moved, with Dr. Wallis seconding, that a tuberculin test of all grade school children be substituted for the present x-rays done in the high school children. Dr. Goodhue moved, with Dr. Fujii seconding, that we continue the x-rays of food handlers and school teachers under 40. Both these measures were passed unanimously.

Dr. Quisenberry then gave a short talk on the traffic accidents being investigated.

The constitution and bylaws were then read by the secretary-treasurer. Dr. Wade, chairman of the Board of Censors, then presented the members selected by the nominating committee. For President, Dr. Wallis; for Vice-President, Dr. Boido; Secretary-Treasurer, Dr. Robert Worth. Censors for the three-year term, Dr. Masunaga; for two-year term, Dr. Boyden; for one-year term, Dr. Wade. Delegate, Dr. Boyden. Alternate, Dr. Cockett. The eleven members present cast their ballots and the above mentioned doctors were unanimously elected.

Dr. Masunaga commended Dr. Peter Kim for his excellent work for the past year as program chairman. The meeting was adjourned at 8:35 P.M. in order to see the film on Heart Disease in Surgery, Pregnancy, and Stress, which was presented by the Upjohn Company.

* * *

The monthly meeting of the Kauai County Medical Society was called to order on Tuesday the 6th of January at 7:30 P.M. at the Wilcox Memorial Hospital. Visitor present was Dr. Zandee.

There was a good deal of discussion concerning the donation to the Hawaii Visitors Bureau. Dr. Kim moved, with Dr. Masunaga seconding, that instead of the Kauai County Medical Society contributing as a whole that each member individually donate whatever he wishes and that a letter be sent to the Hawaii Visitors Bureau notifying them of this decision. The motion was passed eight to four.

VERNON G. BOIDO, M.D.
Secretary

A special meeting was held at Wilcox Hospital Library at 9:30 P.M. following the regular Hospital Staff meeting on January 13, 1959.

Mr. Joseph Veltmann and Mrs. Hilda Inouye of HMSA were present as guests. Mr. Joseph Veltmann discussed Dr. Chock's case now in court in Honolulu. It was pointed out to the Society that there will be an increase in the Surgical Fee Schedule by approximately 50 per cent of the difference between HMSA's present schedule and the Relative Value Schedule which has been adopted by the Honolulu County Medical Society.

Kaiser Medical Plan is competing economically more efficiently at present because surgery procedures are based on higher income clause and payment in full so that patients need not pay doctor out of pocket. Income clause of HMSA to be changed from present one to $7,200, $8,400 $9,600 if each County Society so requests by unanimous vote of members before February 1, 1959. Those of Kauai County Medical Society present at meeting voted unanimously (with one abstention) for a change above in order for HMSA Medical Plan to compete against Kaiser Medical Plan on more equal basis.

P. M. COCKETT, M.D.
Secretary, Pro Tem

Hawaii

The Hawaii County Medical Society held its regular dinner meeting on the evening of November 20, 1958, at 6:30 P.M. Guests present were Drs. J. Warren White, Kaipat, Aniol, Vela Gomez, Dickelmann, Pottinger, and Colonel Ernest Lineberger.

The meeting was called to order at 7:20 P.M. by Dr. Okada who presented Dr. White. Dr. White discussed some bone specimens and orthopedic appliances. Dinner was then served.

At 8:15 P.M. the meeting was again called to order by Dr. Okada. Dr. Helms then moved that Dr. Kenneth Haling's application for membership be accepted. This was seconded by Dr. Steuermann and passed unanimously. Dr. Helms then made a motion that Dr. Harrison S. Paynter, who has retired, be made a life Member of the Society and that in the future his County Society dues be waived. This was seconded by Dr. Crawford and passed unanimously.

Dr. Okada then brought up the matter of the annual December meeting. Dr. Okumoto had declined the appointment of Program Chairman so Dr. Woo was appointed to this chairmanship. It was decided that both the doctors and their wives would attend this meeting, and that the election of next year's officers would be held as a separate portion of this meeting.

Dr. Okumoto of the Operation Hypo Polio Committee expressed his thanks to the doctors who had taken part in this endeavor. Dr. James A. Mitchel gave a report on the last HMSA meeting in Honolulu. Dr. Wipperman gave a report on the Medicare and V.A. meetings in Honolulu. He pointed out that the Honolulu physicians are allowed $4.50 daily whereas we are allowed only $3.00 daily for hospital visits on V.A. patients. He suggested that our secretary write to Dr. Cheim of the V.A. office in Honolulu about these in-

(Continued on page 432)

IN DEBILITATING DISEASE

Patients receiving NILEVAR®

Eat more...
Feel better...
Recover faster

Compared to control patients, those receiving Nilevar (brand of norethandrolone) have repeatedly demonstrated more rapid and more complete recovery from serious acute illness and increased comfort and well-being in chronic illness.

A multitude of case histories are now adding individual clinical color to the earlier controlled investigations which defined the actions of Nilevar as an effective aid in reversing negative nitrogen balance and in building protein tissue.

In typical case reports such gratifying comments as these appear:

Underweight –"Appetite considerably increased within one week. Sense of well-being and vigor increased along with increased appetite."

Prematurity (Birth weight: 2 pounds, 4 ounces) – "Gradual improvement in appetite and capacity for formula. . . . Excellent progress and weight gain for a very immature infant."

Carcinoma of the Uterus –"Within four days appetite became excellent, took full diet. . . . More ambition while on Nilevar. Enjoys life. Takes part in church and other social affairs."

Third Degree Burn–". . . soon began eating all that was offered. . . . Began to show signs of hope for recovery. . . . Perhaps one of the greatest changes was in the appearance of his wounds which were so very much improved."

The dosage for adults is 20 to 30 mg. daily in single courses no longer than three months. For children the daily dosage is 0.5 mg. per kilogram of body weight, in single courses no longer than three months.

Nilevar is supplied in tablets of 10 mg., ampuls of 25 mg. (1 cc.) and Nilevar Drops of 0.25 mg. per drop.

G. D. Searle & Co., Chicago 80, Illinois. Research in the Service of Medicine.

SEARLE

IS THIS YOUR PATIENT?

1.

EARLY POSTMENOPAUSE

Complains of low back pain, vague aches and fatigue

Posture is poor

No x-ray evidence of bone lesions

2.

LATER POSTMENOPAUSE

Back pain is severe, spreading to hips ("girdle pain")

Patient is round shouldered, walks with a stoop

X-ray reveals compression fractures of lower vertebrae

70 AND OVER

Fracture of hip after a minor fall

X-ray reveals fracture of neck of femur

X-ray reveals compression fractures of lower lumbar vertebrae

These three patients have osteoporosis. Early diagnosis and treatment with "Formatrix" is important because osteoporosis is probably the only age change that can be averted. With "Formatrix" therapy, relief from the symptoms of *low back pain, vague aches* and *fatigue* may be obtained in as little as a few weeks. "Formatrix" supplies the essential materials to stimulate increased bone formation and prevent further loss of bone substance that leads eventually to loss of height, stooped posture, and disabling fractures.

The highest incidence of osteoporosis may be found among the 14,000,000 women in the U.S.A. who are 55 years of age and over. Some investigators claim that almost all women past the menopause will show some degree of osteoporosis; furthermore, if all these women were examined carefully, 50 per cent would show x-ray evidence of decreased bone mass.

Suspicion may be the handiest diagnostic tool since presenting symptoms vary from mild to severe and incapacitating pain, and no x-ray evidence of spinal degeneration is available until about 30 per cent of the bone matrix is lost. Between these two extremes there are other signs of estrogen deficiency such as *wrinkled and thinning skin*, a *tendency to appear older than stated years;* there may also be *hypercalciuria* when postmenopausal osteoporosis is complicated by acute osteoporosis of disuse.

Osteoporosis is primarily an atrophic condition of bone matrix formation and any factor that depresses osteoblastic activity or retards the formation of protein and connective tissue such as *prolonged immobilization, cortisone therapy,* or *malnutrition* will favor development of osteoporosis in both male and female.

5878

Ayerst

AYERST LABORATORIES
New York 16, N. Y. • Montreal, Canada

NEW

"FORMATRIX" contains three most essential bone building materials necessary *for matrix* formation, estrogen, androgen and vitamin C.

The estrogen component of "Formatrix" stimulates osteoblastic activity, thus aiding calcium and phosphorus deposition; it also imparts a feeling of "well-being." The anabolic action of methyltestosterone promotes the synthesis of protein and restores a positive nitrogen balance. Together, these hormones have a greater effect on bone and protein metabolism than either alone, and side effects are minimized because of the opposing action of the two steroids on sex-linked tissues. Vitamin C plays an important role in formation of intercellular cement substance and amino acid synthesis. "Formatrix" has a large amount of vitamin C to aid in new bone matrix formation and to further help in the healing of fractures.

"FORMATRIX" – each tablet contains:

Conjugated estrogens equine ("Premarin"®)	1.25 mg.
Methyltestosterone	10.0 mg.
Ascorbic acid	400.0 mg.

Dosage: 1 tablet a day – In the female, three weeks of treatment with a rest period of one week between courses is recommended.

Supplied: Tablets, bottles of 60 and 500.

LITERATURE AVAILABLE ON REQUEST

1.

EARLY POSTMENOPAUSE
No x-ray evidence of bone lesion

2.

LATER POSTMENOPAUSE
X-ray reveals compression fracture of lower vertebrae

3.

70 AND OVER
X-ray reveals fracture of neck of femur

TO RELIEVE LOW BACK PAIN – TO PROMOTE HEALING OF FRACTURES

in osteoporosis

'FORMĀTRIX'

(Brand of Steroid – Vitamin Combination)

for matrix formation

INTER-ISLAND NURSES' BULLETIN

Official Publication of the Nurses' Association, Territory of Hawaii

ROSIE CHANG, *Editor*

FLORA OZAKI, *Associate Editor* — HAZEL KIM, *Associate Editor*

KATSUKO TAKIGUCHI, *Associate Editor* — MILDRED KIM, *Associate Editor*

OLIVE C. PRIDGEN, *Executive Secretary*

President's Message

Have you stopped to think that we may soon have a new name for our organization if Hawaii becomes the fiftieth state? Some of our members attended the meeting called by Governor Quinn to consider what would be done to celebrate the occasion when statehood is proclaimed. We just want you to know that NATH was represented in this forward-thinking affair, emphasizing that we hope to continue to play a part in the progress of our community.

SISTER MAUREEN
President

All of us like to have a voice in affairs affecting us. We wonder if you realize that a major change in our professional organization is under consideration. Your President will represent you in Philadelphia early in May at a meeting of the ANA Advisory Council and representatives of the NLN. The burning question to be discussed was precipitated at the 1958 ANA Convention: *Should we consider a merger of NLN and ANA?*

While we study our local civic status let us also consider what would be most beneficial to Hawaii in our nursing organizations. It is our hope that your thinking will be stimulated and expressed in professional groups so that Hawaii's representative can carry to Philadelphia the conclusions of our group in regard to this proposal.

So many interesting affairs are going on that it keeps us on our toes in trying to keep apace. Legislation affecting nurses on a national basis includes a proposed bill to subsidize building construction and scholarships for basic collegiate programs and for graduates of three-year schools seeking to obtain a degree. We wonder if any consideration is being given to the three-year schools and their needs.

After reading Ruth Sleeper's invigorating article expressing confidence in the education offered and the graduates produced by three-year programs, perhaps we can all take a look at our local programs, and take pride in their status as fully accredited schools. The recent survey of all of our nursing education programs by Miss Donna Monkman from Oregon was presented to the individual schools and also to the Board for the Licensing of Nurses which requested the survey. It was truly a joy to learn of the efforts put forth in every one of these schools of nursing and in hospitals affiliating with these educational programs. This was evident in the practical nurse program, the three-year schools of nursing, and the collegiate program.

We have been reading the unfavorable publicity about hospitals and doctors and nurses and juvenile delinquents that has been presented in the past several months. It is like taking a deep breath of fresh air to realize that the students of today—who will be the leaders of tomorrow—are being very well-prepared to assume their positions in our ranks.

The nursing schools on Oahu need the cooperation of nurses on all islands. In our concern over opportunities for high school graduates, have we overlooked the potential offered in nursing and allied fields? Nurses in every activity and on every island can contribute to the progress of their own profession as well as develop the local economy by taking stock of themselves as they answer this question: What are *you* doing to recruit students for our profession? Remember—in youth is our hope!

Editorials

Statehood

The imminence of the admission of Hawaii as a state in the union requires each of us to understand what statehood will mean—full voting representation in Congress; the right to vote for the president, and to elect state officials, full latitude in lawmaking by the Legislature; governor's, instead of presidential, appointment of judges; elimination of overlapping federal and local authority; equal share in federal grants; a voice in amending the constitution. And all these rights will be irrevokable.

On February 7, one thousand representatives from community organizations met with Governor Quinn in the McKinley High School auditorium to plan for a year-long celebration when Hawaii becomes a state. Tentative proposals and suggestions were outlined and offered for the celebration of the "Year of the Hawaii State." The celebration will be designed to attract the attention of people all over the world.

The Nurses' Association, Territory of Hawaii, and the District of Oahu were represented at this meeting and nurses will be kept up to date with information on celebration plans as it is released to us.

Workshop on Publications and Public Relations

A workshop on the theories and practices involved in presenting information to people, the first of its kind, was held at Mabel Smyth Lounge on December 13, 1958. Participants included all members of the Committee on Nursing Information.

Since news about nurses and their activities usually are entered on the Women's Page, Mrs. Drue Lytle, Women's Editor of the Honolulu Advertiser, had much to offer in her presentation of the "Good News Story." A survey shows us that women "shop" the paper. A typical reader scans:

1. Page 1
2. Women's page for women; sports page for men
3. Comics
4. Editorials
5. Letters to editors

Mr. Byron Feldman, account executive with Holst and Male, Inc., spoke on "Advertising and the Small Newsletter." The information we received on layout, advertising, effective writing, and publishing formula will be especially helpful in improving our Newsletter.

In the afternoon, we heard Mr. Kuff, TPA executive, on "Public Relations," the art of communication for better understanding. A thought provoking suggestion made by Mr. Kuff was: "Why not consider HNA (Hawaii Nurses Association) as our association name to prepare ourselves for statehood?"

A Salute to Kauai Nurses

The Garden Isle boasts 55 regular and 15 associate members and we are proud to feature them in this issue of the Inter-Island Nurses' Bulletin. In the following articles you will read of the projects the Kauai District Nurses' Association, a small but active group known for their contributions to the community in which they live. Our thanks to Patricia McGuire for covering her island's activities.

District and Section News

KAUAI

PRESIDENT: Miss Elvie Manley, Wilcox Memorial Hospital, Lihue, Kauai

SECRETARY: Mrs. Eunice Sun, Box 546, Kalaheo, Kauai

DATE OF MEETING: First Monday of the month.

TIME: As announced.

PLACE: As announced.

On January 21, 1959, a group of nurses from Mahelona Hospital, and the Health Department met with Mrs. Giles, of the Tuberculosis Association, and Mrs. Jenkins, of Wilcox Hospital, at the Training Center on Kauai for a review of First Aid.

Through the efforts of Miss Annajean Ray, American Red Cross Representative on the Island of Kauai, we were fortunate to have Mr. Roger C. Plaisted speak to us. Mr. Plaisted is the Director of Safety Services of the Los Angeles Chapter of the American Red Cross in California.

He gave an up-to-date review of first aid, discussed the care of wounds and burns, explained and demonstrated the various methods of artificial respiration, showed the methods of applying a bandage, and supervised a group when they prac-

ticed techniques in moving the wounded. This afforded the nurses an opportunity to participate through actual practice and stimulated questions. Indeed, the group came away with a better insight as to what first aid really is, "doing when you have to with what you have."

* * *

Misses Yong Hi Chung and Hyo Jin Chung, two nurses from Korea, were recent visitors to Kauai. They attend the University of Hawaii and are working for their certificate in Public Health Nursing. It was our pleasure to show them the various activities of the Public Health Nurse in the community. They plan to return to Korea where the skills learned in Hawaii will be used to better the health of the people of their country.

* * *

A newcomer to Wilcox Hosptial is Miss Flora Billings, R.N., R.R.L. She is a graduate of Wyoming General Hospital and has recently arrived from Idaho. Miss Billings is the Medical Librarian and Assistant Administrator at Wilcox.

Another new member, Miss Herminia Marquez, is with the Department of Health as a public health nurse for the Kapaa District. A 1958 graduate of Loretto Heights College in Colorado, she is originally from Kauai.

* * *

Tuberculin skin testing is underway in eight of Kauai's schools. Half of the schools are being tested this year and the remaining half will be tested next year.

Mrs. Evelyn Giles from the Tuberculosis Association on Kauai provides educational material to the schools on tuberculosis and the skin test. The public health nurses assist the school in planning the physical set-up for skin testing. The school in turn plans for parent and student participation.

Kauai High School was the first school to be tested this year. Due to very good cooperation the testing ran smoothly. Approximately 800 students were given skin tests. All positive readings are referred for a chest x-ray when the x-ray unit comes to the school. Thus affording good follow through.

* * *

Mr. and Mrs. Paul Matsunaga are the proud parents of their first child, daughter Dorene. Mrs. Matsunaga was the former Arline Minatoya.

* * *

The Kauai District Nurses' Association held its first meeting of the year on January 5, 1959, at the Hanapepe Health Center.

The members voted to raise the Association dues to $23.50 a year. Annual reports were read by officers and committee chairmen.

Newly elected officers are: President, Miss Elvie Manley (re-elected); Vice-President, Miss Myrna Campbell; and Treasurer, Mrs. Jahne Hupy; Board of Directors, Mrs. Miyoko Ednaco and Mrs. Nellie Hiyane. Holdover officers are: First Vice-President, Miss Edna Chapman; Secretary, Mrs. Unus Sun; Board of Directors, Mrs. Evelyn Giles and Miss Edith Hinchliffe.

Two new arrivals to Kauai are Blanche Stradeski and Lorraine Bridger. They are 1955 graduates of St. Michael's Hospital in Toronto, Canada. Both have been working at Providence Hospital in Anchorage, Alaska. At present they are employed at Kauai Veterans Memorial Hospital.

Introducing Our Kauai Correspondent

MISS McGUIRE

A newcomer to our Inter-Island Bulletin staff is Patricia Kaonohilani McGuire. Likened to our American version of "you are the apple of my eye," the melodious sounding Kaonohilani in Hawaiian means "you are the pupil of my eye." Another interpretation of Kaonohilani is "the throne of heaven."

Of Caucasian-Hawaiian-Chinese background "Pat," as she is called by friends, was born in Honolulu and attended Sacred Hearts Academy. She received her high school education at Notre Dame Convent in Alameda, California. After a year at San Francisco Junior College, Pat received her R.N. title in 1952 from Highland School of Nursing in Oakland, California.

The only daughter of a family with three boys, Pat worked on the staffs of St. Francis and Shriners' Hospitals. Sponsored by a Honolulu Chamber of Commerce scholarship, Pat completed the requirements of a course leading to public health nurse's certificate. The district she now has is in Lihue, Kauai, and we hear Pat is loving the work there.

Outside of work, Pat keeps herself busy with gardening as she enjoys to work with plants. The beach is another area you can see Pat often as she likes swimming as a pastime. Wilcox Memorial Nurses' Residence is "home away from home" for Pat.

Welcome to our staff, Pat, and we hope to hear from you often as you represent the Kauai district members on the Inter-Island Nurses' Bulletin.

BYLAWS

of

Nurses' Association, Territory of Hawaii, Inc.

(as Revised February 15, 1959)

ARTICLE I—Title and Functions

Section 1. The name of this association shall be the Nurses' Association, Territory of Hawaii, Inc.

Section 2. The functions of the Nurses' Association, Territory of Hawaii, Inc. shall include the following:

(a) To assist with the definition of the functions of nurses and to promote standards of professional nurse practice.

(b) To assist with the definition of the qualifications for the practitioners of nursing including those in the various nursing specialties.

(c) To promote legislation and to speak for nurses in regard to legislative action concerning general health and welfare programs.

(d) To promote and assist with periodic surveys of territorial nursing resources.

(e) To promote and protect the economic and general welfare of nurses.

(f) To provide, interpret and promote professional counseling service to individual nurses, and to their employers in regard to employment opportunities and available personnel.

(g) To cooperate with the Hawaii League for Nursing in activities which concern both organizations.

(h) To represent nurses and serve as their territorial spokesman with allied professional and government groups and with the public.

(i) To establish standards for nurses professional registries and approve registries which meet state standards.

ARTICLE II—Constituent Associations

Section 1. District nurses' associations which have been or which hereafter may be organized, whose constitution and bylaws are in harmony with the bylaws of this association and have been approved by majority vote of the board of directors of the association, shall be recognized as constituent associations of the Nurses' Association, Territory of Hawaii, Inc.

Section 2. The boundaries of the district nurses' associations shall be clearly defined and recorded by the board of directors of this association. The boundaries may be changed by two-thirds vote of the board of directors of this association provided such change has been approved by each district nurses' association involved.

Section 3. Any district nurses' association which fails to comply with the requirements of these bylaws or for other causes deemed sufficient, may be disqualified as a constituent association of the Nurses' Association, Territory of Hawaii by two-thirds vote of the board of directors provided due notice has been given to the constituent nurses' association three months before the vote is taken.

Section 4. A district nurses' association which has been disqualified may be reinstated by two-thirds vote of the board of directors of this association.

ARTICLE III—Membership

Section 1. The membership of this association shall consist of the active and associate members of the constituent associations.

Members of district nurses' associations shall be nurses who are graduates of state accredited schools of professional nursing offering programs of not less than two years of instruction and clinical practice in hospitals and in other community agencies, each of whom has been granted a license to practice as a registered nurse in at least one state, which license has not been revoked in any state for professional misconduct.

Section 2. The active members of this association shall be the active members of the constituent associations and shall have all privileges of membership. Only active members shall have the privileges of voting in sections and serving as delegates at meetings of this association and the American Nurses' Association. Only active members shall be eligible to hold office and to serve as chairmen of standing committees.

Section 3. The associate members of this association shall be the associate members of the constituent associations. Only members who do not anticipate employment in nursing during the current calendar year may be associate members. Upon such employment, associate members shall be required to transfer to active membership, failing which they shall forfeit the rights of associate membership and be stricken from the rolls by the constituent association of which they are members.

Associate members shall have all of the privileges of membership except those of voting, serving as delegates, holding office or serving as chairmen of standing committees. No member or applicant for membership shall be required to become an associate member if active membership is preferred.

Section 4. The presentation to this association of a classified typewritten list of members in good standing in the constituent nurses' association, signed by the secretaries of those associations, together with the annual dues of such members, shall establish such members of constituent nurses' associations as members of this association.

Section 5. Every member in good standing in the Nurses' Association, Territory of Hawaii is a member of the American Nurses' Association and every member has representation through that association in the International Council of Nurses.

Section 6. A member of another constituent association of the American Nurses' Association who moves to this territory may transfer membership to this association without further payment of dues for the remainder of the fiscal year, provided such nurse meets the requirements for membership in this association and the request for transfer of membership is accepted. The request for transfer shall be sent to the executive secretary of the state nurses' association issuing the transfer.

A member of this association who moves out of this territory may apply to the secretary of this association for transfer of membership to the state nurses' association of the state of new residence.

Section 7. A member may transfer from one district nurses' association to another within the territory without further payment of dues for the remainder of the fiscal year, provided such nurse meets the requirements for membership of the association in the new district and the request for transfer of membership is accepted by that association. Application for transfer of membership shall be made to the secretary of the constituent nurses' association of which the nurse is currently a member.

Section 8. Honorary recognition may be conferred by a unanimous vote at any annual convention on persons who have rendered distinguished service or valuable assistance to the nursing profession, the name or names having been recommended by the board of directors. Honorary recognition shall not be conferred on more than two persons at any annual convention.

ARTICLE IV—Dues

Section 1. (a) The annual dues to this Association shall be twenty dollars and fifty cents ($20.50) per capita for active members, payable on or before February 10 by the constituent associations which includes seven dollars and fifty cents ($7.50) per capita dues to the American Nurses' Association.

(b) The annual dues for associate members of this Asso-

ciation, shall be three dollars and fifty cents ($3.50) per capita payable on or before February 10 by the constituent associations, which includes two dollars ($2.00) per capita dues to the American Nurses' Association.

Dues for associate members who become active members during the fiscal year and who have paid dues as associate members for that year, shall be seventeen dollars ($17.00) in addition to the three dollars and fifty cents ($3.50) previously paid. From the amount seventeen dollars ($17.00) paid in, five dollars and fifty cents ($5.50) will be sent to the American Nurses' Association.

(c) The dues for nurses who graduate and are licensed to practice professional nursing after July 1 of any year and become active members of this Association shall be ten dollars and twenty-five cents ($10.25) per capita, for that same year, and payable by the constituent association which includes three dollars and seventy-five cents ($3.75) per capita dues to the American Nurses' Association.

Section 2. Dues received by district nurses' associations after February 10 shall be paid monthly to this association during the remainder of the year.

Section 3. District nurses' associations which have not paid dues for any members to this association by March 15 shall be notified by the treasurer, and those which have paid no dues by April 15 shall be disqualified as constituent associations of the Nurses' Association, Territory of Hawaii.

Section 4. Not later than March 15 the Treasurer of this Association shall pay to the American Nurses' Association seven dollars and fifty cents ($7.50) per capita for the active membership and two dollars ($2.00) per capita for the associate members of this Association for the current calendar year.

Section 5. Dues received by this association after March 15 shall be paid to the American Nurses' Association monthly during the remainder of this year.

Section 6. All dues paid to the American Nurses' Association shall be accompanied by a typewritten classified list of the members for whom dues are paid.

ARTICLE V—Officers

Section 1. The officers of this association shall be a president, a first vice president, a second vice president, a secretary, a treasurer and six (6) directors.

Section 2. Officers shall be elected at annual conventions as hereinafter provided.

Section 3. Officers shall perform the duties usually performed by such officers and also such duties as are specified in these bylaws and designated by the Board of Directors of this association.

Section 4. Vacancies in office shall be filled as hereinafter provided.

Section 5. No officer shall serve more than two (2) successive terms in the same office. Service for a portion of the term greater than half shall be considered a term.

Section 6. No person shall be elected to serve as an officer or director of this association, who at the same time, would be serving as a section chairman.

ARTICLE VI—Duties of Officers

Section 1. (a) The president shall preside at all meetings of this association and all meetings of the board of directors and be a member ex-officio of all committees except the Committee on Nominations.

(b) The president shall deliver an annual address and perform all duties of the office.

(c) The president or her alternate shall serve as a representative of the association at meetings of the Advisory Council of the American Nurses' Association.

(d) In the even years the president shall serve as chairman of the Coordinating Council of the Nurses' Association, Territory of Hawaii, Inc. and the Hawaii League for Nursing.

(e) The president shall be empowered to appoint all standing and special committees, except Nomination Committee, of the association with the approval of the board of directors.

Section 2. In the absence of the president, the vice-presidents in order of rank shall assume the duties of the president. They shall also assume other duties as assigned to them by the board of directors. In the event of a vacancy occurring in the office of president, the first vice-president shall assume the duties of president until the next annual convention or until a successor is elected. In the event of a vacancy occurring in the office of first vice-president, the second vice-president shall assume the duties of the first vice-president until the next annual convention or until a successor is elected. The first vice-president shall be a member ex-officio of the Committee on Constitution and Bylaws.

Section 3. The duties of the second vice-president shall be the same as those of the first vice-president and she shall serve as temporary parliamentarian in the absence of the first vice-president. She shall be a member ex-officio of the Committee on Nursing Information and Membership.

Section 4. (a) The secretary shall be responsible to keep minutes of all meetings of this association, and the board of directors.

(b) She shall be the custodian of the seal of this corporation and the permanent files of same.

(c) She shall be a member ex-officio of the Committee on Nominations.

(d) She shall prepare and deliver to the annual convention of this association, a report on the accomplishments of this association during the preceding calendar year.

(e) She shall deliver to her successor within one month after her election to office all records and the seal of this association.

(f) She shall notify all officers, directors and committees of election or appointment; send notices of the time and place of all meetings; send to the secretary of the American Nurses' Association the names and addresses of all officers of the Nurses' Association, Territory of Hawaii, Inc. immediately after their election.

(g) Conduct the general correspondence of the association and the board of directors.

(h) She shall keep a correct list of the names and addresses of all members of this association.

The executive secretary shall assume such duties in connection with the work of the secretary as shall be designated by the board of directors.

Section 5. (a) The treasurer shall receive and have charge of all funds of the association, deposit such funds in a bank designated by the board of directors and pay such bills only as shall have been approved by the president.

(b) She shall report to the board of directors the financial standing of the association whenever requested to do so and make a full report to the association at each annual convention.

(c) She shall keep an itemized account of all receipts and disbursements and give a written report at meetings of the board of directors and of the association.

(d) She shall give a bond subject to the approval of the board of directors, and submit all books of account for audit as specified by the board of directors.

(e) She shall pay dues to the American Nurses' Association and send notice to district associations as specified in Article IV, Dues, of these bylaws.

The treasurer shall be a member of the Committee on Finance.

The retiring treasurer shall deliver to her successor within one (1) month after her election to office all money, vouchers, books and papers of the association in her custody with a supplementary report covering all transactions from the date of last audit to the date of surrendering her accounts to her successor.

The executive secretary shall assume such duties in connection with the work of the treasurer as shall be designated by the board of directors.

After the board approves attendance at any meeting called by the American Nurses' Association and for which that association will pay travel expenses but for which this association must pay living expenses, the treasurer may advance payment for plane fare and living expenses at the rate of $15 per day.

Section 6. All officers except the secretary and treasurer upon expiration of their term of office shall surrender all property of this association in their possession pertaining to their respective offices to the newly elected president.

Section 7. In addition to the duties of the officers set

forth herein, the officers shall have such other duties as implied by their titles.

ARTICLE VII—Elections

Section 1. The president, the second vice-president and the treasurer shall be elected at the annual conventions held in the even years; and the first vice-president and the secretary shall be elected at the annual conventions held in the odd years.

The term of office of these officers shall commence at the adjournment of the annual convention at which they are elected and shall continue for two (2) years or until their successors are elected.

Section 2. At each annual convention two (2) directors shall be elected to serve for three (3) years or until their successors are elected.

Section 3. All elections shall be by secret written ballot. A ticket of at least two (2) candidates for each office shall be prepared and presented to the convention assembled by the Committee on Nominations. No names shall be presented to the convention unless the nominee has agreed to serve if elected.

Section 4. A plurality vote of those present, entitled to vote and voting, shall constitute an election.

In case of a tie, the choice shall be decided by lot.

Section 5. On the first day of the annual convention the president shall appoint a special committee of tellers who shall act also as inspectors of the election. There will be one teller from each constituent nurses' association. No nominee or candidate for any office shall be appointed a member of this committee.

Section 6. The secretary of this association shall furnish the tellers at least two (2) hours before the opening of the polls a complete list of delegates and alternates of constituent nurses' associations entitled to vote. The teller in charge of the list of delegates shall check the names of those voting.

Section 7. The teller in charge of the ballot box shall place her official mark upon the back of each ballot. The voters shall then place the ballot in the ballot box.

Section 8. Polls shall be open for such periods of time as specified by the board of directors and noted in the program of the annual convention.

Section 9. Additional nominations may be made from the floor of the convention with the consent of those being nominated.

ARTICLE VIII—Meetings

Section 1. This association shall hold an annual meeting known as the Annual Convention of the Nurses' Association, Territory of Hawaii, Inc. at such time and place as shall be determined by the board of directors.

Section 2. The order of business of each annual convention of this association shall be in accordance with the program adopted at the beginning of the convention and shall include:

a. Call to order
b. Invocation
c. Appointment of tellers
d. President's address
e. Roll call of delegates
f. Report of Program Committee
g. Reading of minutes
h. Reports of officers
i. Reports of sections
j. Reports of district nurses' associations
k. Reports of standing committees
l. Reports of special committees
m. Old business
n. New business
o. Election of officers
p. Adjournment

Section 3. Special meetings of this association may be called by the board of directors and shall be called by the president upon written request of a majority of the district associations.

Section 4. Notice of all meetings of this association shall be sent to the president and the secretary of each district association and to all members of this association prior to the meeting in question. Notices of the annual convention shall be mailed at least one (1) month before the first day of the convention and notices of special meetings shall be mailed at least ten (10) days before the first day of the meeting.

Section 5. Members of the Hawaii Student Nurses' Association may attend meetings of the Nurses' Association, Territory of Hawaii, Inc.

Section 6. Meetings of the Hawaii Student Nurses' Association may be held in connection with annual conventions at such times as shall be designated in the program of the conventions.

ARTICLE IX—Representation

Section 1. The voting body at all meetings of this association shall consist of the accredited delegates in attendance from the district nurses' associations.

Section 2. Each district nurses' association shall be entitled to one delegate for each ten (10) active members, or portion thereof, computed on the basis of membership thirty (30) days prior to the opening day of the annual convention of the Nurses' Association, Territory of Hawaii, Inc. Any district nurses' association having less than ten (10) active members shall be entitled to one delegate. (Care should be taken in the district associations to choose delegates from the various fields of nursing.)

Section 3. The secretary of each district nurses' association shall send a list of accredited delegates from that association by the most expeditious means, to the secretary of the Nurses' Association, Territory of Hawaii, Inc., at least thirty (30) days before the opening of the convention. A list of alternates will also be submitted by each district nurses' association in conjunction with the list of accredited delegates.

Section 4. If all the accredited delegates from the district nurses' association are not in attendance at the convention, the president of the district nurses' association, or her empowered representative, may fill the vacancies from the list of alternates for that association. If the list of alternates is exhausted and vacancies remain, the president of the district association, or her empowered representative, shall be empowered to appoint as delegates other members of that association who are in attendance.

Section 5. All delegates shall present credentials at the time of registration.

Section 6. Each accredited delegate shall be permitted to cast not more than one (1) vote in any election or any matter coming before the convention. This provision applies also to alternates or members who are acting as delegates appointed to represent a district nurses' association.

Section 7. (a) The Nurses' Association, Territory of Hawaii, Inc. is entitled to representation at biennial conventions or special meetings of the American Nurses' Association on the basis of one (1) delegate for every two hundred (200) active members of each section or fraction thereof, such delegate or delegates to be elected in a manner to be determined by the section. Each section, through the Nurses' Association, Territory of Hawaii, Inc., shall certify the names and addresses of the delegate or delegates and their respective alternates elected for such section. The number of delegates to be computed on the number of active members of each section of the Nurses' Association, Territory of Hawaii, Inc. in good standing in the ANA on December 31 of the year preceding a biennial convention or special meeting. Each section of each district nurses' association shall be asked to submit to the secretary of the corresponding territorial section a list of nominees for delegates and alternates to the biennial convention or special meeting. The number of delegates to be nominated by each section of the district shall be determined on the basis of membership as stated above. If any district nurses' association has no sections, it may submit names from the various fields of nursing within the district on the basis of membership as stated above. If such a district has less than one hundred (100) members, it may submit the name of one (1) nominee for delegate.

(b) Since the Nurses' Association, Territory of Hawaii, Inc. is entitled to three (3) delegates-at-large to biennial conventions or special meetings of the American Nurses' Association, these delegates-at-large shall be selected by the

NATH board of directors from names submitted by the districts.

Section 8. If a special meeting is held in the month of January or February, for the purpose of computing the number of delegates to which any section of the Nurses' Association, Territory of Hawaii, Inc. shall be entitled, the number of active members shall be deemed to be the active members in good standing as active members of the American Nurses' Association on November 1 of the year preceding a special meeting, as evidenced by dues paid to this association or postmarked by such time.

Section 9. The representatives of this association at meetings of the Advisory Council of the American Nurses' Association shall be two (2) active members of this association, one (1) of whom shall be the president of this association or her alternate.

ARTICLE X—Board of Directors

Section 1. The members of the board of directors of this association shall be eleven (11) composed of: the president, first vice-president, second vice-president, secretary, and treasurer, and six (6) directors, four (4) of whom shall be selected, one each from the respective districts, to serve as the official representative of that district, in addition to these the chairmen of organized sections of the Nurses' Association, Territory of Hawaii, Inc., shall be regular members of the board of directors.

Section 2. The regular meeting of the board of directors shall be held immediately preceding and immediately following each annual convention* at the place where the annual convention is held. The regular meetings of the board may also be held at such time and place as shall from time to time be determined by action of the board.

Section 3. Special meetings of the board of directors may be called by the president of this association upon seven (7) days' notice to each member of the Board either personally or by mail or by telegraph and shall be called by the president in like manner or by like notice upon the written request of five (5) members of the board of directors. Special meetings shall be held at such time and such place as may be specified in the notice thereof.

Section 4. In the intervals between regular meetings of the board of directors, the president of the association may refer and submit by mail or telegraph to the members of the board of directors definite questions relating to the affairs of the association which, in the opinion of the president, require immediate action on the part of the board of directors. The result of such a referendum which requires a majority vote of the personnel of the board of directors shall control the action of the association, its board of directors, officers, sections.

Section 5. Officers of the Territorial Student Nurses' Association shall be eligible to attend meetings of the board of directors of this association in rotation, one officer attending each meeting but without vote.

ARTICLE XI—Duties of the Board of Directors

Section 1. The board of directors shall:

(a) Transact the general business of the association in the interim between annual conventions.

(b) Establish major administrative policies governing the affairs of the association and devise and mature measures for the association's growth and development.

(c) Provide for the maintenance of territorial headquarters and for making such office the center of activities of the association, including such work of the officers and committees as may be deemed expedient; provide for the proper care of materials, equipment and funds of the association, for the payment of legitimate expenses and for the annual auditing of all books of account by our agent if agency agreement is in effect; otherwise audit shall be made by a certified public accounting firm.

(d) Report to the association assembled at each annual convention through the secretary of this association the business transacted by the board during the preceding year.

(e) Assume responsibility with regard to constituent district nurses' associations as specified in Article II of these bylaws.

(f) Appoint an executive secretary and other personnel, define their duties and fix their compensation.

(g) Create special committees as the need for them arises, dissolving said committees upon the accomplishment of their missions.

(h) Approve the appointment of all members to standing and special committees made by the president in accordance with these bylaws.

(i) Have power to fill any vacancies on the board of directors except a vacancy occurring in the office of the president or first vice-president.

(j) Decide upon the exact date and place for holding the annual convention and provide for the payment for a place of meeting when necessary; decide upon the time and place for meetings of the advisory council; hold meetings of the board of directors as hereinbefore provided.

(k) Provide for the establishment and dissolution of sections in accordance with these bylaws.

(l) Verify referendum votes of the board of directors.

(m) Appoint the association's representatives on Board of Management of the Mabel Smyth Memorial Building Committee in the manner provided for by the agreement made on October 15, 1939 between the Nurses' Association, Territory of Hawaii, Inc., the Hawaii Medical Association and The Queen's Hospital.

(n) Recommend to the Governor of the Territory the names of persons suitable for appointments to the Board for the Licensing of Nurses when vacancies on said board occur.

(o) Shall have the right and authority by unanimous vote to appeal to the membership of this association, when necessary, for supplemental finances to carry on the program of the association.

(p) Appoint an active member, in addition to the president, or her alternate, to represent this association at meetings of the Advisory Council of the American Nurses' Association.

(q) Have the power to employ legal counsel and such other paid personnel as may from time to time be needed at fees made known to the board of directors in advance of such employment.

Section 2. No individual member of the board of directors shall pledge any property of this association for security either for a personal or an association loan nor will members contact any liabilities in the name of the association without first having in their possession written permission from the treasurer of this association as approved by the board of directors.

Section 3. There shall be an executive committee, called the sub-NATH, of the board of directors composed of the president, the officers and board members located on the Island of Oahu, and one other island director to be selected in rotation. This committee shall have all the powers of the board of directors to transact business of an essential nature between board meetings. All transactions of this committee shall be verified at the next regularly scheduled meeting of the board of directors.

ARTICLE XII—Standing Committees

Section 1. The following standing committees except the Committee on Nominations shall be appointed at or immediately after each annual convention to serve until the next annual convention or until their respective successors are appointed:

1. Committee on Finance
2. Committee on Program
3. Committee on Arrangements
4. Committee on Legislation
5. Committee on Constitution and Bylaws
6. Committee on Nominations (elected)
7. Committee on Membership

* Except where government regulations or conditions incident upon war may render this impossible.

8. Committee on Nursing Information
9. Committee on Margaret Jones Memorial Fund
10. Economic Security Committee

Section 2. Standing committees except the Committee on Nominations may be composed of both active and associate members of the association and shall assume such duties as are specified in these bylaws and such other duties as may be assigned by the board of directors. Only active members shall be chairmen of standing committees.

Section 3. The Committee on Finance shall consist of at least five (5) members and not more than ten (10), one of whom shall be the chairman as appointed by the president with the approval of the board of directors. The treasurer of the Nurses' Association, Territory of Hawaii, Inc. shall be a member of said committee. This committee shall prepare a budget for the year, advise as to expenditure of funds and report the same to the board of directors at meetings of that body.

Section 4. The Committee on Program shall consist of at least three (3) members representing at least the three major fields of nursing. This committee shall prepare a program for all meetings of this association, subject to the approval of the board of directors. This committee shall act in an advisory capacity to Committee on Program of district nurses' associations throughout the year.

Section 5. The Committee on Arrangements shall consist of at least three (3) members. This committee shall work in conjunction with the Committee on Program, subject to the approval of the board of directors, and shall make local arrangements for the annual convention and perform such other duties as shall be designated by the board of directors.

Section 6. The Committee on Legislation shall consist of at least five (5) members. The membership of this committee shall be representative of all major branches of nursing and various geographic sections of the territory.

The duties of this committee shall be to:

Study the program and objectives of the association to determine the needs for territorial legislation.

Receive proposals for legislative action from other committees of the association.

Evaluate all proposed legislation relating to nursing and the programs of the association in order to determine and recommend the goals and resources of the association.

Devise methods for effective legislative action.

Section 7. The Committee on Constitution and Bylaws shall consist of at least three (3) members. This committee shall receive all proposed amendments to the bylaws of this association. It shall be responsible for securing an opinion from the American Nurses' Association Committee on Constitution and Bylaws with regard to the same, and shall recommend proposed action thereon to the board of directors of this association. Such proposed amendments shall be submitted for action to the voting body at the annual convention of the association in accordance with the provisions for amendments in these bylaws. This committee shall review the constitution and bylaws of any district nurses' association wishing to become a constituent association of the Nurses' Association, Territory of Hawaii, Inc. The committee shall report its findings to the board of directors of this association, whose decision as to the acceptability of the district shall be final. This committee shall advise district nurses' associations concerning proposed amendments to their constitution and bylaws in order that these may be kept in harmony with the bylaws of this association and of the American Nurses' Association. These first vice-president shall be a member ex-officio of this committee.

Section 8. The Committee on Membership shall consist of three (3) or more members representative of various geographic areas of the territory. This committee shall cooperate with the ANA Committee on Promotion of American Nurses' Association Membership, and shall devise ways and means of cooperation with the district nurses' associations in securing members and in methods of organization for making such membership effective. Such action shall be subject to the approval of the board of directors. The second vice-president shall be a member ex-officio of this committee.

Section 9. The Committee on Nominations shall consist of not less than three (3) active members. Not more than one (1) member of this committee is to be a member of any one district nurses' association. Ninety (90) days prior to the annual meeting the Committee on Nominations shall send to the district nurses' associations the names of officers then serving, and those whose terms of office will expire at the next annual convention, and shall request from each district nurses' association a list of names of members qualified to fill vacancies in office and willing to serve if elected. These members should be representative of various fields of nursing service. Such lists shall be signed by the president or secretary of the respective district nurses' associations and shall be submitted by the district nurses' associations to the Committee on Nominations of this association sixty (60) days prior to the annual convention. From these lists the Committee on Nominations shall prepare a ticket consisting of at least two (2) nominees for each office to be filled. This ticket shall include representatives of major branches of nursing and various geographic sections of the territory. The secretary shall be a member ex-officio of this committee.

Nominations Committee is to be an elected committee.

Section 10. The Committee on Nursing Information shall be representative of the various nursing groups in the territory. It shall be the function of this committee to plan and promote the territorial nursing information program subject to the approval of the board of directors. The committee shall inform nurses about the activities of the professional nursing organizations so that they will support the organizations, benefit by their services and participate intelligently in their programs. Also, the committee shall promote better understanding of professional nursing by the public so that it will make discriminating use of professional nursing service and insist on sound preparation for nursing. The second vice-president shall be a member ex-officio of this committee.

Section 11. The Committee on Margaret Jones Memorial Fund shall consist of at least three (3) members. The vice-president shall be a member (ex-officio) of this committee. The chairman of this committee, as appointed by the president, should be a nurse who has previously served as a member of the committee. It shall be the functions of this committee to receive requests for loans or gifts and to act upon them in accordance with the policies established in the deed of trust. To recommend to the board of directors, when the need arises, that certain loans be written off the books as gifts. To meet with representatives of the Bishop Trust Company each year following the audit of the fund to evaluate the yearly performance of our investments.

Section 12. Hawaii Student Nurses' Association members may be eligible to attend committee meetings of this association.

Section 13. The Committee on Economic Security shall consist of all seven (7) NATH section chairmen and a prorated number from each section. This prorated number will allow one (1) economic security committee member for every ten (10) members of each section. The section chairmen will not be counted in this ratio. The chairman of this committee shall be selected by the President.

The purposes and functions of this committee shall be to—

1. Furnish opportunity for committee members to discuss mutual problems and recommend group plans to the Board.
2. Furnish opportunity for clearance of activities and progress being made in each section.
3. Collect facts pertinent to the economic security program desired by the section.
4. Study minimum conditions of employment as established by sections in order to coordinate one with another.
5. Study minimum conditions of employment as established by sections and make recommendations to the Board for acceptance or further study.
6. Review survey questionnaires to be used by sections, if desired.

ARTICLE XIII—Sections

Section 1. (a) Sections may be established by a majority vote of the board of directors of this association.

(b) A section may be established for each of the following groups of professional nurses:

(1) Private duty nurses
(2) General duty nurses
(3) Public health nurses
(4) Institutional nursing service administrators
(5) Educational administrators, consultants, and teachers
(6) Occupational Health nurses
(7) Special groups

(c) Members of a branch group in the territory shall be eligible to apply for status as a section when they meet the following criteria:

(1) In the Nurses' Association, Territory of Hawaii, Inc. with a membership of less than 1500, if they number at least thirty-five (35); and
(2) If the members of the branch group desiring status as a section have functioned as a conference group within one of the established sections for a year; and provided the number of members of the section does not fall below the numerical criterion for establishment of the section (35).
(3) If they have demonstrated group interest; and
(4) If they have developed a program; and
(5) If the needs of such a group cannot be met in any existing section or by continuation as a conference group.

(d) Membership in the Special Groups Section of the Nurses' Association, Territory of Hawaii, Inc. shall be open only to those nurses who are not eligible for membership in any other section.

Section 2. Functions of sections may include the following:

(a) Define the qualifications for membership which are consistent with the general membership requirements of the Nurses' Association, Territory of Hawaii, Inc.

(b) Make rules for its government, provided these shall in no way conflict with the bylaws of the Nurses' Association, Territory of Hawaii, Inc. and shall be approved by the board of directors.

(c) Define the functions, standards, and qualifications for practice within the occupational field, these to be developed for each special field by the practitioners within it.

(d) Initiate studies or experiments for the improvement of practice within the field in relation to the overall purpose of the Nurses' Association, Territory of Hawaii, Inc.

(e) Study the general welfare and economic needs of the members and develop desirable standards of employment.

(f) Promote the organization of subunits within the section in order that groups which have like interests shall have the opportunity of meeting to consider the economic security program separately from other groups in the same section whose economic interests might be somewhat different. The subunits shall have the privilege of reporting directly to the board of directors of the Nurses' Association, Territory of Hawaii, Inc.

(g) Represent the occupational interests in district, territorial, and national meetings.

(h) Develop relationships with allied professional groups for conferences or committee work related to the objectives of the Nurses' Association, Territory of Hawaii, Inc.

(i) Conduct programs of special interest to the members of the occupational group or participate with other sections that have similar interests.

(j) Organize conference groups on request for special interests within the section.

(k) Develop and actively promote a program for intergroup relations within the section.

(l) Plan a program of work and prepare an appropriate budget annually for presentation to the Nurses' Association, Territory of Hawaii, Inc. Finance Committee.

(m) Make pronouncements in its own name, provided they are not in opposition to the policies accepted by the House of Delegates and do not purport to represent the policies of the association as a whole.

(n) Interpret all policies accepted by the House of Delegates that affect the section and publish same in its own name.

(o) Keep open direct channels of communication between the Nurses Association, Territory of Hawaii, Inc. and district sections, with information going simultaneously to the executive secretary of the Nurses' Association, Territory of Hawaii, Inc. and the presidents and the secretaries of the district nurses' associations.

Section 3. Officers of the section shall be chairman, vice chairman and a secretary. The officers shall be elected annually at the annual convention of this association. The newly elected secretary will forward immediately to the secretary of this association the names and addresses of all newly elected officers.

Section 4. A section may be dissolved by two-thirds vote of the board of directors upon the following conditions:

Upon the recommendation of the section concerned, or upon agreement by the board of directors and the section that there is no reason for its continuance, or upon the section's failure to carry out its objective or to conform with the requirements or principles of this association.

Section 5. A section which has been dissolved by action of the board of directors of this association in which the section does not concur, may appeal to said board of directors for reconsideration, or may appeal to this association at any annual convention. The decision of this association shall be final.

Section 6. All members of a section in attendance at a section meeting are entitled to vote on all matters coming before the section.

Section 7. Sections may engage in fund-raising, provided however, that plans for fund-raising be submitted to and approved by the board of directors.

ARTICLE XIV—Duties of Constituent Association

Section 1. It shall be the duty of each constituent district nurses' association of the Nurses' Association, Territory of Hawaii, Inc.:

(a) To require that all of its members have the qualifications enumerated in Article III, Section 1, of these bylaws.

(b) To send to the secretary of this association the names and addresses of all officers of the district nurses' association immediately following their election or appointment.

(c) To confer with the Committee on Constitution and Bylaws of this association before adopting any proposed amendments to its constitution and bylaws.

(d) To send to the secretary of this association a copy of the constitution and bylaws and all amendments adopted by the district nurses' association.

(e) To pay dues to this association as provided in Article IV, Sections 1 and 2, of these bylaws. Such dues shall be sent to the treasurer of this association with typewritten classified lists in duplicate of the members for whom dues are paid.

(f) To send to the secretary of this association the names and addresses of all those who are entitled to attend the annual convention as members of the voting body at least thirty (30) days before the opening day of the convention and to inform the secretary of all changes as promptly as possible.

(g) To nominate delegates to the biennial convention and special meetings of the American Nurses' Association in conformity with Article IX, Sections 7 and 8, of these bylaws and to send the names of those nominees, with addresses, to the secretary of this association.

(h) To report to this association as may be required by the board of directors in order to comply with the constitution and bylaws of the Nurses' Association, Territory of Hawaii, Inc.

ARTICLE XV—Coordinating Council

Section 1. Membership. There shall be a Coordinating Council which shall be composed of all the officers and other members of the board of directors of Nurses' Association, Territory of Hawaii, Inc. and all of the officers and other members of the board of directors of the Hawaii League for Nursing.

Section 2. Officers. Starting with the president of the Nurses' Association, Territory of Hawaii, Inc., the president of that association and the president of the Hawaii League

for Nursing shall serve alternately for one (1) year as chairman of the Coordinating Council.

Section 3. Purpose and Functions. The coordinating Council shall promote the coordination of those programs that are of common concern to the Hawaii League for Nursing and the Nurses' Association, Territory of Hawaii, Inc. To promote such coordination, the Coordinating Council shall:

(a) Serve as a forum for the discussion of different points of view for the purpose of reaching agreement when feasible;

(b) Plan together, serve as a clearing house for activities of common concern to both the Hawaii League for Nursing and the Nurses' Association, Territory of Hawaii, Inc., and agree on allocation of new major programs; and

(c) Consider priorities for and timing of interrelated activities of the Hawaii League for Nursing and the Nurses' Association, Territory of Hawaii, Inc.

The Coordinating Council shall also act as sponsors of and advisers to the state student nurses' council or organization.

Section 4. Steering Committee. There shall be a Steering Committee for the Coordinating Council which shall be authorized to make recommendations when, because of an emergency or other special situation, a recommendation must be made before the Coordinating Council can meet. The Steering Committee for the Coordinating Council shall be composed of the president and elected secretary of the Nurses' Association, Territory of Hawaii, Inc. and the president and elected secretary of the Hawaii League for Nursing.

Section 5. Special Committees. The Coordinating Council of the Nurses' Association, Territory of Hawaii, Inc. and the Hawaii League for Nursing shall have authority to appoint special committees if necessary.

ARTICLE XVI—Quorum

Section 1. Seven (7) members of the board of directors, one (1) of whom shall be the president or a vice-president, and representatives from a majority of the constituent district nurses' associations, shall constitute a quorum for the transaction of business at any annual convention or special meeting of the Nurses' Association, Territory of Hawaii, Inc.

Section 2. Seven (7) members of the board of directors, one (1) of whom shall be the president or a vice-president, shall constitute a quorum at any meeting of the board of directors.

Section 3. A majority of members of the Advisory Council shall constitute a quorum of the Council.

Section 4. A majority of members of any standing or special committee shall constitute a quorum.

ARTICLE XVII—Fiscal Year

The fiscal year for this association shall be the calendar year.

ARTICLE XVIII—Parliamentary Authority

The rules contained in *Robert's Rules of Order Revised* shall govern all meetings of the board of directors, committees, special meetings and conventions of this association in all cases wherein they are applicable and not inconsistent with these bylaws.

ARTICLE XIX—Official Organs

The American Journal of Nursing and the Inter-Island Nurses' Bulletin shall be the official organs of this association.

ARTICLE XX—Amendments

Section 1. These bylaws may be amended at any annual convention of this association by two-thirds vote of the accredited delegates present and voting. All proposed amendments shall be in possession of the secretary of this association at least thirty (30) days before the date of the annual convention and shall be appended to the call for the meeting.

Section 2. These bylaws may be amended at any annual convention by unanimous vote without previous notice.

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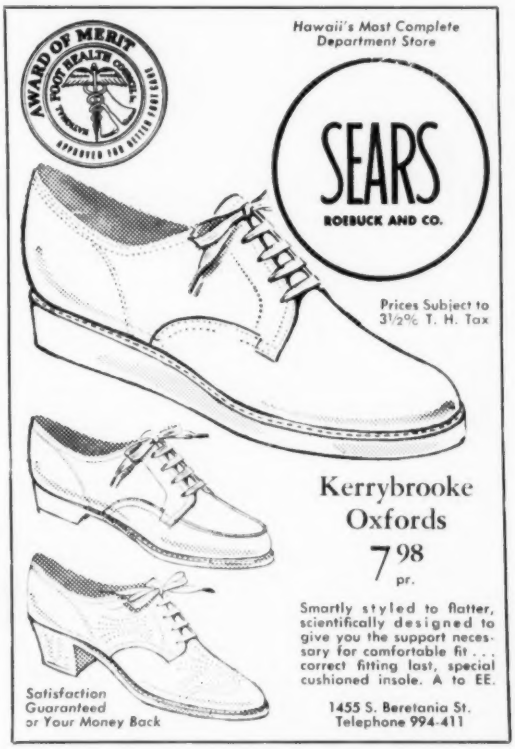
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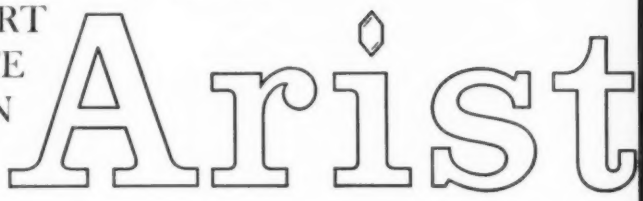

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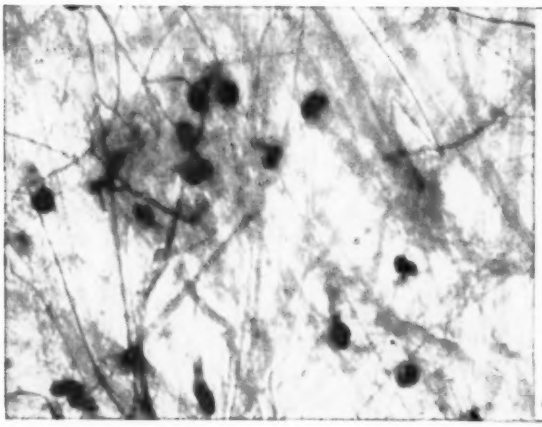

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HAWAII TECHNOLOGISTS' BULLETIN

Official Publication of the Hawaii Society of Medical Technologists

LYDIA C. MARTENS, *Editor* MUN FOOK SHINN, *Associate Editor*

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HSMT President Writes

DEAR MEMBERS OF HSMT:

The year 1958-1959 should be especially memorable for us since it will mark the 10th anniversary of our Hawaii Society of Medical Technologists.

We have seen during the past years the progress of our Society with an increase in membership and its recognition in the medical field. The HAWAII MEDICAL JOURNAL for the first year now also serves as our Society's official bulletin. The first successful seminars have been initiated as part of our educational program to keep us informed of the latest developments in laboratory medicine.

It was most gratifying to see the interest shown by so many of you at our first seminar meetings. We hope to see many more of you at the remaining seminars planned. Read the schedule of the topics to be discussed and be sure to attend and contribute to the discussions.

Plan now to attend the 10th Annual Convention of our Hawaii Society of Medical Technologists, tentatively set for April 23, 24. Our co-chairmen for this convention, Mrs. Dorothy Matsuo and Miss Edith Eckstein, and their committee will be making this an outstanding event. An impressive list of speakers and topics, including workshop sessions, will be chosen that will be certain to arouse your interest and curiosity. Don't miss this convention—there will be many surprises in store for you! You will receive more details by letter.

A handful of interested members have been working hard in order to present you with a stimulating and educational program. May I ask the rest of you for your cooperation and support? Not until we have the help of each registered medical technologist, will we ever be able to have a professional organization to be proud of. Our Society urgently needs you. Let us all give our wholehearted support now!

CLARA YUEN

HSMT Seminar Schedule

Ann Stegmaier, Chairman of the Education Committee, announces the following seminars to be held at the Blood Bank of Hawaii Conference Room.

March: The Application of Tissue Culture in Virology by Clara Yuen, The Queen's Hospital.

April: Blood Bank Procedures and Techniques by Ann Stegmaier, Blood Bank of Hawaii.

ASCP and CAP Convention Notes

The 37th Annual Meeting of the American Society of Clinical Pathologists (ASCP), and the 12th Annual Meeting of the College of American Pathologists (CAP) convened in Chicago during the first week in November, 1958. The scientific program included many subjects which would be of interest to you, but I would like to devote most of this space to recent economic developments in the profession of medical technology in which you are so vitally interested.

As you know a National Committee for Careers in Medical Technology was formed in 1954 sponsored by your Society, the American Society of Medical Technologists (ASMT), as well as by the ASCP and CAP, to initiate a program designed to increase enrollment in AMA approved schools of medical technology. The original financing came from the three societies, but since then most of the funds have been received from outside sources, including the American Cancer Society and various foundations, in a ratio of about four to one. Two films have been produced which have been shown by most of the nation's television stations, and information has been disseminated by other means to students in colleges and high schools throughout the country. As a result there are at the present time 30 per cent more students registered in approved schools than in 1954, when the committee was established, and there are now 462 approved schools. Three possible areas for future efforts by the NCCMT were said to be the development of a national scholarship program, sponsorship of a pilot program involving use of an educational director to coordinate and improve medical technology education, and sponsorship of a comprehensive study of the needs in medical technology training programs.

You probably also know that as of 1962, three years of academic college work will be required for admission to approved schools of medical technology. This requirement was finally adopted by the American Medical Association last June upon the recommendation of your Society. At present 66 per cent of the 452 approved schools are affiliated with colleges and the training period counts toward a baccalaureate degree.

The Board of Schools of Medical Technology approved the ASMT resolution that teaching supervisors in approved schools have a baccalaureate degree and three years of teaching experience, and that a study be conducted to determine curriculum revisions in view of the pending three-year college requirement.

The Board of Registry reported that general inquiries increased 100 per cent during 1958, indicating much greater interest in the profession, and the Registry appropriated $4,000.00 for use by the ASMT in developing state and district seminars for continuing education.

There were indications in the Assembly meeting of the College, and the Councilor's meeting of the ASCP that salary scales are improving rapidly throughout the country, most often through joint efforts of pathology and technology societies, which have established committees to confer with hospital administrations and hospital associations. This improving income picture is no doubt responsible at least in part for the increased enrollment in AMA approved schools. In general, I believe the outlook for your profession is brighter now than it has been for some time.

I. L. TILDEN, M.D.

Tech Tips . . .

Spending too much time cleaning those used blood slides?

Tired of having buckets of soaking slides all over the lab?

Hear! Hear! Try dropping the slides, one at a time, into a pan of one part alcohol, one part water, and two parts green soap. After soaking for two to five minutes, a gentle wipe with the finger tips, a rinse in running water, and those messy things are ready to be drained, alcohol rinsed, and polished. It is even easier than it sounds and doesn't stain the hands.

P.S. Some people like to add a little detergent such as Alconox or Hemo-sol, to the soap solution.

* * *

Tired of spending half an hour to stain a bone marrow? Want more contrast in the stains for your micrographs? We found the answer in the stain described below and, after using it almost a year, still like it.

To make one liter of stain—

GRIND TOGETHER:	3.0 grams Wright's stain 0.3 grams Giemsa's stain 30 cc glycerin
DISSOLVE:	3.2 grams Na_2HPO_4 1.6 grams KH_2PO_4
INTO:	970 cc Absolute Methyl Alcohol

Add the buffered alcohol to the stain-glycerin mixture, shake on a shaking machine 20 minutes, and let stand 24 hours. Filter before using. Stain blood film for 15 seconds and dilute with distilled or tap water for 15-30 seconds. Bone marrow requires about twice as much time with both stain and diluent.

P.S. As in everything else, the better the ingredients, the better the end product.

We use dyes by National Aniline.

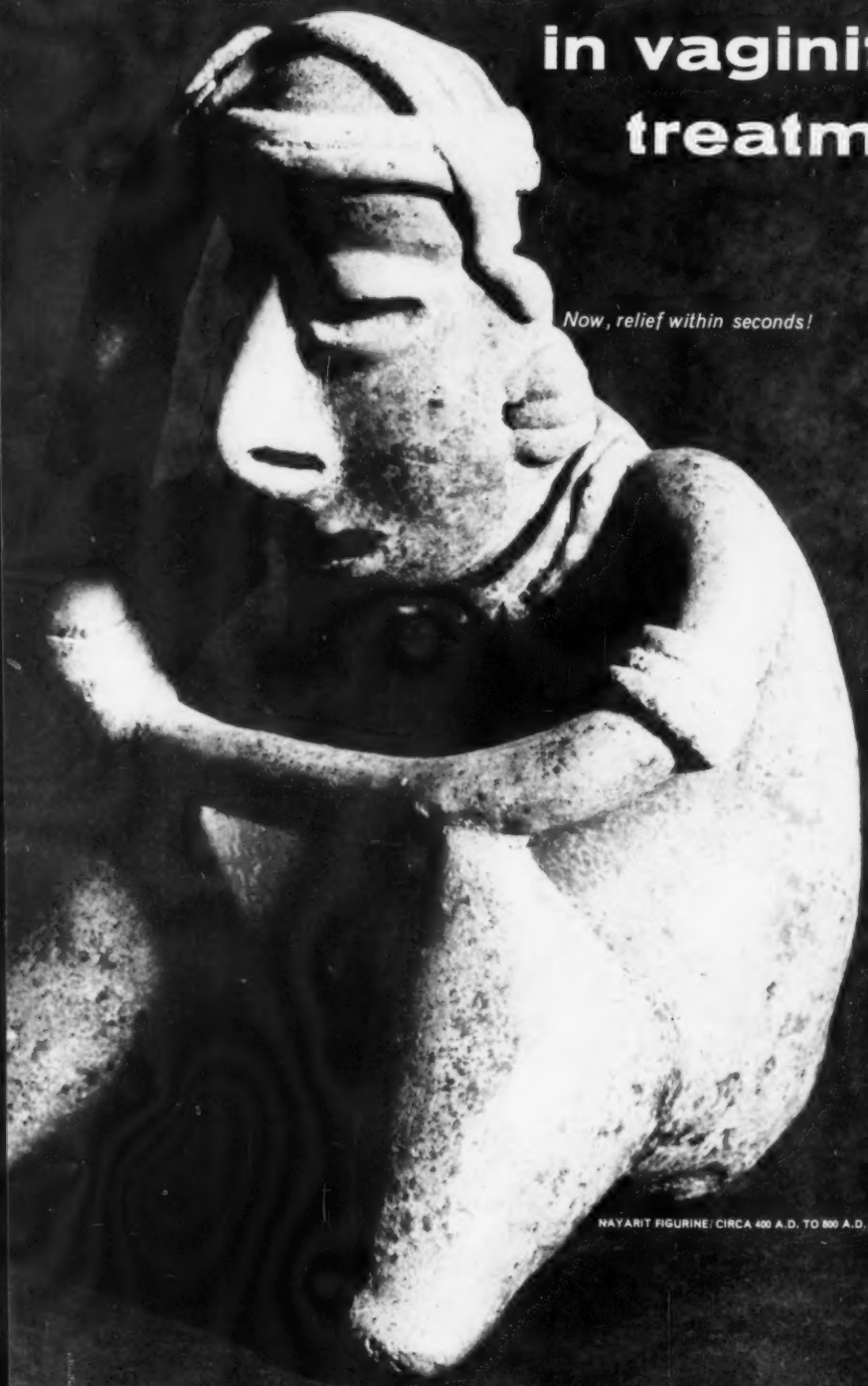
V. J. COEN, M.T.(ASCP)

Triva

Triva's "Chelating Agent" Intensifies Organism Disintegration Comparative newcomers to the field of therapeutics...are the Chelating Agents. Though effective in minute quantities and non-toxic, these agents combine with calcium, phosphorus and other metallic ions to form stable, extractable compounds. / Triva's Chelating Agent* attacks the metallic ions in the cell walls of vaginitis organisms...rendering them more susceptible to the germicidal activity of Triva's surface active agents. / Within seconds after her first douche, your vaginitis patient gets relief from intense itching, burning and other symptoms. Within 12 days, most cases of trichomonal and non-specific vaginitis are rendered organism-free (Monilia genus may require longer treatment). / Administration: Douche, b.i.d., for 12 days. *Supplied: Package of 24 individual 3 Gm. packets. Composition: 35% Alkyl Aryl sulfonate (wetting agent and detergent); 5% Di-sodium ethylene bis-iminodiacetate (chelating agent); 53% Sodium sulfate; 2% Oxyquinoline sulfate; 9.5% dispersant. / *Di-sodium ethylene bis-iminodiacetate.*

BOYLE & Company / Los Angeles 54, Calif.

major breakthrough in vaginitis treatment



Now, relief within seconds!

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Cadillac's dignity and bearing, its every sculptured-in-steel contour and every touch of chrome, stainless steel or brushed aluminum has been skillfully designed to achieve an enduring and timeless motor car. Powered by a spectacular high-performance engine and smooth responsive Hydra-Matic transmission, Cadillac assures you superb riding comfort, important stability and handling ease . . . and marvelous economy of ownership. Its dependability, safety, spaciousness and luxurious beauty are unprecedented.

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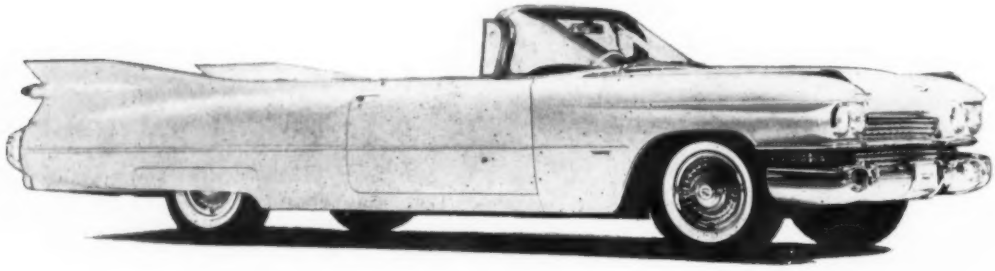

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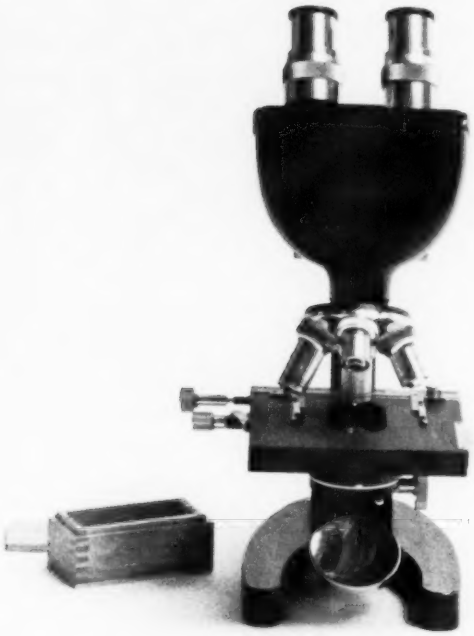
for the control of all coccal infections



ABBOTT'S ANTIBIOTIC TRIAD

against staph-, strep- and pneumococci

After Millions of Prescriptions
An Unparalleled Safety Record

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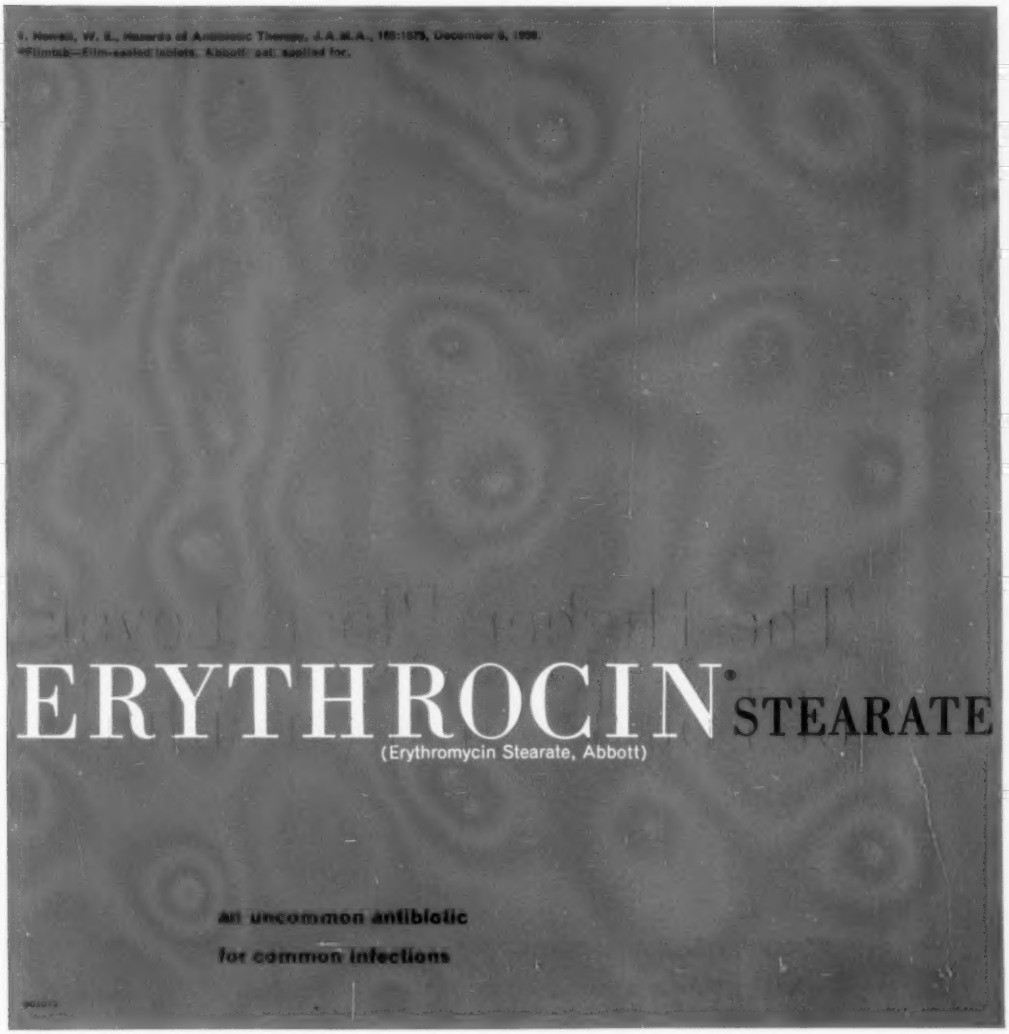
Provides fast, high blood and tissue concentrations—Because ERYTHROCIN Stearate is rapidly absorbed, patients get therapeutic blood and tissue levels within 30 minutes—and effective concentrations for at least six hours.

Supported by an unparalleled safety record—During all the years ERYTHROCIN has been prescribed, serious reactions have been practically nonexistent. Unlike penicillin, allergy is no problem. And, in contrast to "broad spectrum" action, the normal intestinal flora is virtually unaltered with ERYTHROCIN therapy. And only recently, a well-known investigator said, "Erythromycin is by far the least toxic of the commonly used antibiotics.[1]"

Offers bactericidal action—Unlike broad-spectrum antibiotics, ERYTHROCIN is classed as a bactericidal agent. It offers lethal action against common coccic invaders—resulting in prompt clinical responses.

Provides convenient dosage forms—Usual adult dose is 250 mg. four times daily. Children's dosage is reduced in proportion to body weight. ERYTHROCIN comes in Filmtabs* (100 and 250 mg.), bottles of 25 and 100. Also, in oral suspension and for intramuscular and intravenous use. Abbott


1. Herrell, W. E., Hazards of Antibiotic Therapy, J.A.M.A., 165:1575, December 6, 1958.
*Filmtab—film-sealed tablets. Abbott pat. applied for.
ERYTHROCIN STEARATE
(Erythromycin Stearate, Abbott)
An uncommon antibiotic
for common infections

for those penicillin-sensitive organisms

The Higher Blood Levels of Potassium Penicillin V

COMPOCILLIN-VK Indications—Against all organisms sensitive to oral penicillin therapy. For prophylaxis and treatment of complications in viral conditions. As a prophylaxis in rheumatic fever and in rheumatic heart disease.

COMPOCILLIN-VK Dosage—Depending on the severity of the infection, the usual adult dose is 125 mg. to 250 mg. (200,000 to 400,000 units) every four to six hours. For children, dosage may be reduced in proportion to body weight.

COMPOCILLIN-VK Supplied—In Filmtabs, 125 mg. (200,000 units), bottles of 50 and 100; 250 mg. (400,000 units), bottles of 25 and 100. For oral solution, COMPOCILLIN-VK comes in 40-cc. and 80-cc. bottles. When reconstituted with water, each appealing (it's a clear red solution) 5-cc. teaspoonful represents 125 mg. (200,000 units) of potassium penicillin V.

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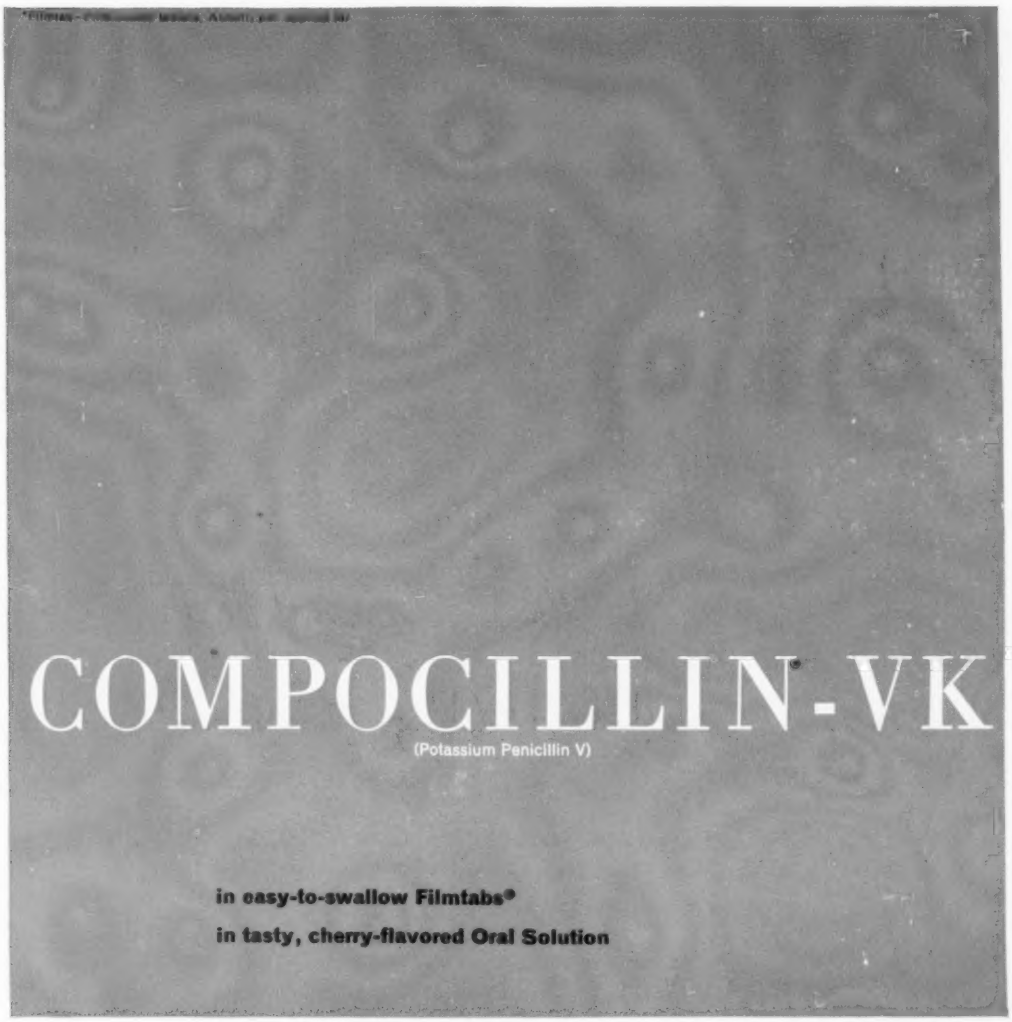

against serious and resistant coccal infections

An Important Lifesaving Antibiotic

The dramatic story of SPONTIN can never really begin to be told.

In little more than a year, this potent antibiotic has compiled an incredible record for saving lives —and often, after all other therapy had failed. Majority of successes involved patients critically ill with staphylococcal infections—conditions that had resisted all other known antibiotic therapy.

Meanwhile, careful attention to dosage recommendations has practically eliminated toxicity and side effects as serious obstacles to therapy. Also, recent improvements have been made in the manufacture of SPONTIN; the drug is now made from pure crystals.

So far, SPONTIN has proved to be a good answer, perhaps the best answer to the resistant staphylococcal problem—and of real value in other serious coccal infections.

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1. Sixth Annual Symposium on Antibiotics, Washington, D. C., Oct. 15, 16, 17, 1958.

Crystallized

SPONTIN®

(Ristocetin, Abbott)

Prepared from pure crystals

Provides Outstanding Clinical Effectiveness Against Coccal Infections, Including Resistant Staphylococci and Enterococci[1]

Provides Bactericidal Action Against Coccal Infections[1]

HAWAII MEDICAL ASSOCIATION

(Continued from page 385)

AMA'S QUESTIONS OF FREE CHOICE:

The questions posed by the AMA on the matter of free choice of physicians were read by Dr. Bergin. It was explained that we had received a bulletin asking that the opinion of our members be reported to the AMA sixty days before the next meeting. A lengthy discussion followed and it was felt that the questions as asked were unanswerable. It was also determined that we had received no direct letter from the AMA on this.

ACTION:

Dr. Nishigaya moved, seconded by Dr. Cushnie, that we not poll our members on this matter. The motion carried.

CANADIAN RETIREMENT PLAN:

Mr. Kennedy was asked to talk on this. He said they had received information from Dr. Blair of Canada and that Honolulu had tried to work out a savings plan but they had run into all kinds of difficulty with the SEC. He said he expects to receive more information from Dr. Blair and would report to the Council again on this matter.

RECORDING OF MINUTES:

The advisability of recording the meeting was next discussed.

ACTION:

Dr. Nishigaya moved, seconded by Dr. Cushnie, that this matter be tabled for the present. The motion carried.

AMA DELEGATE'S REPORT:

The members of the Council adjourned to a smaller room where Dr. Bergin read the report of Dr. Arnold. There was considerable discussion again about the questions which the AMA wants the constituent societies to answer relative to closed panel medicine. However, there were no comments on the other portions of the report.

ACTION:

Dr. Burden moved, seconded by Dr. Nishigaya, that the report be accepted and placed on file. The motion carried.

OFFICIAL SEAL:

Dr. Bergin said that the Honolulu Star-Bulletin Printing Company drew up the design and charged us something over $60.00 for the art work. It was explained that the bill was settled for $25.00 and had been paid. There was considerable criticism of the design. Dr. Bergin suggested that perhaps we could offer a prize, not necessarily monetary, at the annual meeting for the design of a seal. It was explained that it had been suggested that the JOURNAL might get a new cover and that the seal could be used on this as well as on our letterheads, etc.; that we are probably the only association without an official seal. The Council felt that the cover of the JOURNAL was very good as it is.

Dr. Bergin said we wouldn't use the seal for anything except display purposes.

ACTION:

Dr. Yap moved, seconded by Dr. Burden, that we table the matter of an official seal. The motion carried.

DUES:

Dr. Bergin read the memorandum about the several financial matters. The matter of waiver of dues was discussed, whether or not it should be automatic, depending upon the county's action.

ACTION:

Dr. Spencer moved, seconded by Dr. Nishigaya, that the Council accept the county societies' recommendations on the waiver of dues and also on the granting of life memberships. The motion carried.

Mr. Kennedy brought up the matter of partially waived dues. It was pointed out that our bylaws state we will waive dues only when they have been completely waived by the county. Dr. Spencer recommended that this be brought up before the House of Delegates.

PREMARITAL BLOOD TEST:

Dr. Bergin said that Dr. Reppun had written a lengthy letter and that he had suggested we recommend that the premarital blood test be abolished principally because of the decline of syphilis. It was noted that the Advisory Committee on Venereal Disease had met to discuss this matter and had made a report. They recommended that the abolishment of the test not be considered at this time. Dr. Cushnie said that the question in his mind was what does this examination include. He had patients come to him that had gone to other doctors who had refused to sign the slip because the patient wouldn't submit to a pelvic and that is the problem he would like to have cleared up. Dr. Richert thought the whole idea of the blood test was a means of putting teeth in the so-called gin law so that a person can't get a license while he's loaded and with the three-day waiting period it is convenient to require some sort of physical examination. Dr. Spencer said he didn't see how we could gain anything by even bringing it up. Dr. Cushnie wanted to know if the doctor is liable if he didn't do a physical examination on one of these cases. Why leave ourselves open to a possible libel suit some day. He said he believed in premarital Wasserman tests. Dr. Bergin read the complete report of the Venereal Disease Committee and asked why we didn't accept their recommendations.

ACTION:

Dr. Spencer moved, seconded by Dr. Burden, that we should, through our Legislative Committee, ask some kind of interpretation from the President of the Board of Health or the Attorney General regarding the extent of our responsibility in doing premarital examinations. The motion carried.

Dr. Spencer further recommended that when we get the opinion it should be published in the JOURNAL.

TRANSFER OF COMPONENT SOCIETY MEMBERSHIP:

Dr. Bergin read a letter from the Honolulu County Medical Society which outlined a conflict between their bylaws and those of the HMA. It was pointed out that this problem has been referred to the Judicial Council of the AMA. Dr. Burden wondered if it wouldn't be a matter to be referred to the Constitution and Bylaws Committee. It was pointed out that Honolulu wanted an opinion from the Council. Mr. Kennedy said there was an additional letter which he had written to the AMA, not the copy of the one that was distributed, asking them about denying membership to doctors and requesting them to cite any cases which had been taken to court and advising of the outcome.

(Continued on page 418)

In the Treatment of Rheumatic Disorders Greater stability of maintenance dosage minimizes risks of hormonal imbalance

In Sterazolidin, the anti-inflammatory actions of prednisone and Butazolidin* are combined to permit lower effective dosage of each. Clinical experience has indicated that patients can be well maintained on this combination over prolonged periods with relatively low, stable dosage levels of each component, thus minimizing the problems arising from excessively high doses of corticosteroids. Other side effects have also been gratifyingly few. Antacid and spasmolytic components are contained in Sterazolidin capsules for the benefit of patients with gastric sensitivity.

Sterazolidin®: Each capsule contains prednisone 1.25 mg.; phenylbutazone 50 mg.; dried aluminum hydroxide gel 100 mg.; magnesium trisilicate 150 mg.; homatropine methylbromide 1.25 mg.

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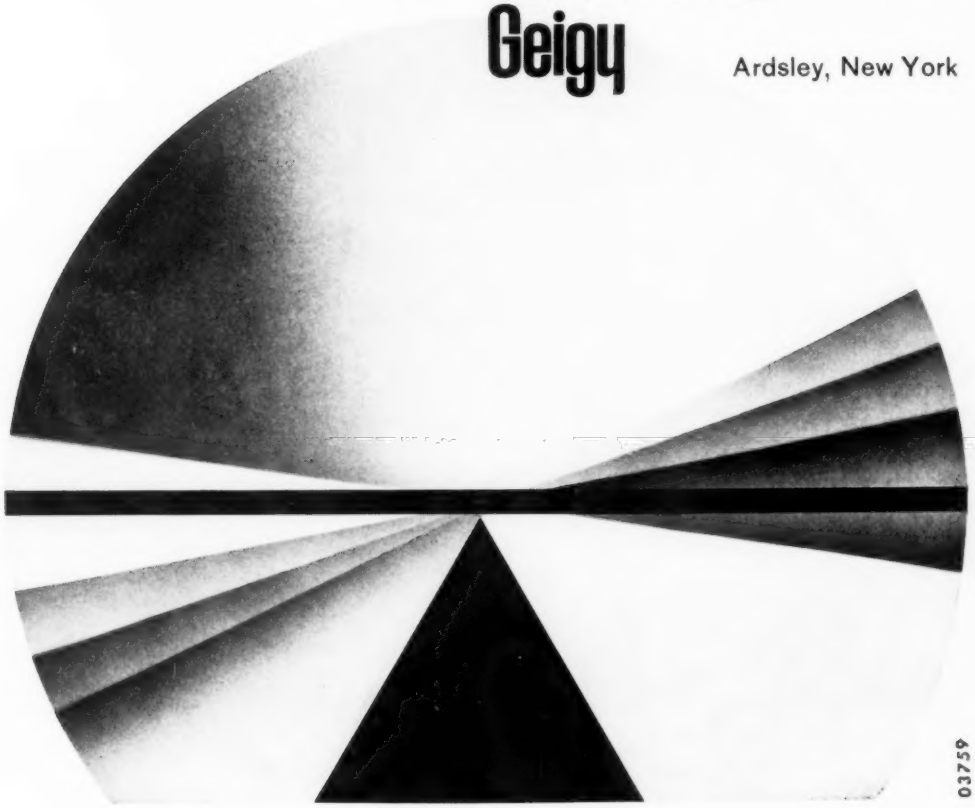

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- Relieves "incessant itching" and inflammation
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- Helps restore normal acid mantle
- ***Rarely sensitizes***

Contains: 'Aerosporin'® brand Polymyxin B Sulfate, Neomycin Sulfate, and Hydrocortisone (free alcohol) in a sterile, slightly acid, aqueous suspension.

Available in dropper bottles of 5 cc.

for infected and inflamed ears

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- Counteracts "sogginess" of ear canal
- Eradicates *Pseudomonas* and other common causes of otitis
- Hygroscopic
- Antifungal for *Monilia* and *Aspergillus*
- Helps restore normal acid mantle
- ***Rarely sensitizes***

May be used prophylactically in patients with recurring "swimmer's ear."

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Available in dropper bottles of 10 cc.

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HAWAII MEDICAL ASSOCIATION

(Continued from page 414)

ACTION:

Dr. Burgess moved, seconded by Dr. Nishigaya, that we defer action on this matter until we hear from the county society on what they have heard from the AMA Judicial Council. The motion carried.

BUDGET:

The budget, which was reviewed while the Council was meeting with Hawaii County, was brought up for approval.

ACTION:

Dr. Burgess moved, seconded by Dr. Mizuire, that the budget be approved as proposed with the deletion of the allotment for the roster. The motion carried.

REPORTING OF COMMITTEE PROCEEDINGS:

It was felt that it is up to the President and Chairmen to decide how to handle the reporting of the proceedings and whatever they work out will be satisfactory.

BYLAWS AND PARLIAMENTARY COMMITTEE:

Dr. Bergin read a letter from Dr. Ando which contained certain recommendations. After a brief discussion, the consensus was that if any changes are to be made, the Bylaws and Parliamentary Committee could recommend these changes when they make their revisions.

ACTION:

Dr. Nishigaya moved, seconded by Dr. Burgess, that the Bylaws and Parliamentary Committee be made a standing committee and be given authority to revise the present bylaws. That the recommended revision be referred to the Council when it is completed. The motion carried.

The meeting adjourned at 10:30 P.M.

NOTES AND NEWS

(Continued from page 387)

All members of the profession are cordially invited to attend and are urged to make arrangements as soon as possible if they wish to be assured of adequate facilities.

An outstanding scientific program by leading surgeons promises to be of interest to all doctors. Nine surgical specialty sections are held simultaneously.

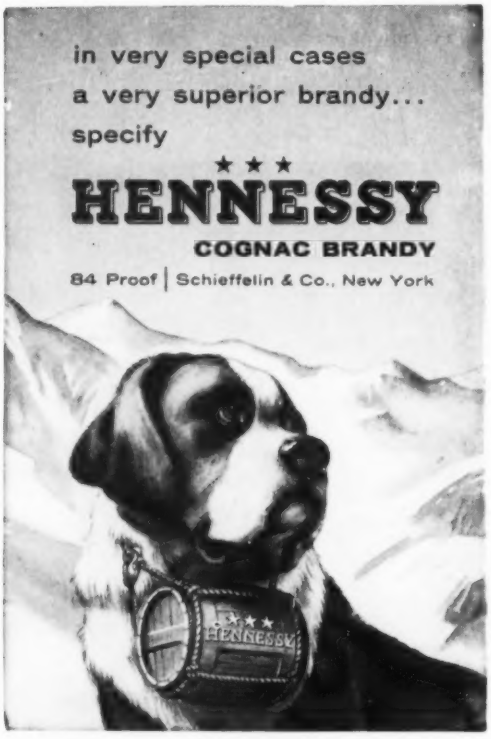
Further information and brochures may be obtained by writing to Dr. F. J. Pinkerton, Dirctor General of the Pan-Pacific Surgical Association, Suite 230, Alexander Young Building, Honolulu 13, Hawaii.

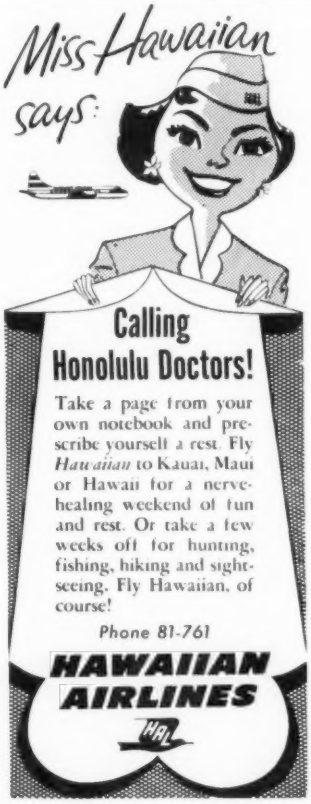
Kimble Methodology Research Award

Nominations for the Eighth Kimble Methodology Research Award are being accepted until June 1, 1959. The cash award of $1000 and a silver plaque will be presented at the annual meeting to be held in Atlantic City in October.

The candidate's work to be considered for nomination should be either (1) a fundamental contribution which serves as a baseline for development of diagnostic methods which fall within the province of the public

(Continued on page 422)

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COMPARE in milliequivalents per liter (mEq/L):

	Baxter Isolyte	*Normal Blood Plasma*
Sodium	140	140
Potassium	10	5
Calcium	5	5
Magnesium	3	3
Chloride	103	103
Acetate	47*	–
Citrate	8*	–
Bicarbonate	–	27

ISOLYTE contains in each 100 cc.:

Sodium Chloride U.S.P. 0.50 Gm.; Potassium Chloride U.S.P. 0.075 Gm.; Calcium Chloride U.S.P. 0.035 Gm.; Magnesium Chloride Hexahydrate 0.031 Gm.; Sodium Acetate N.F. 0.64 Gm.*; Sodium Citrate U.S.P. 0.075 Gm.*

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JUST ONE TABLET DAILY

provides therapeutic sulfa levels for 24 hours...
Highly soluble in acid and alkaline media... rapidly absorbed, producing fast, effective plasma-tissue concentrations sustained for the entire day. Simple, single 0.5 Gm. daily dose minimizes patient dosage confusion. At least equivalent to 4 to 6 Gms. daily of previous sulfonamides. Does not produce renal complications.[1]

with low incidence of sensitivity reactions...
KYNEX is extremely low in toxic potential.[2,3] Cutaneous or other objective sensitivity reactions are rare, as demonstrated in a large scale evaluation of clinical toxicity.[2] Also minor subjective reactions are less likely to develop when the recommended dosage is used.[2]

Dosage: Adults, 0.5 Gm. (1 tablet) daily following an initial first-day dose of 1 Gm. (2 tablets).

TABLETS, 0.5 Gm., Bottles of 24 and 100.

also available—KYNEX Acetyl Pediatric Suspension, cherry-flavored, 250 mg. sulfamethoxypyridazine activity per teaspoonful (5 cc.). Bottles of 4 and 16 fl. oz.

1. Editorial, New England J. Med. 258:48, 1958.
2. Vinnicombe, J.: Antibiotic Med & Clin. Ther. 5:474, 1958.
3. Sheth, U. K., et al.: Ibid., p. 604, 1958.

for improved control
WHENEVER SULFAS ARE INDICATED

KYNEX®

Sulfamethoxypyridazine Lederle

Lederle LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

NOTES AND NEWS

(Continued from page 418)

health laboratory, or (2) the adaptation of a fundamental contribution to make it of use in a diagnostic laboratory.

Nominations may be made by the authors, their associates, or by others.

Academy of General Practice Scientific Congress in Hawaii

Dr. Philip Thorek of Chicago will be the principal speaker for the 1959 Invitational Scientific Congress in Hawaii. Category I credit will be given for attendance at the three scientific sessions which will be held at the Matson Meeting House on April 13 and 14. These will be breakfast sessions convening at 7:30 A.M. followed by scientific lectures. In addition there will be an evening meeting at Mabel Smyth Auditorium at 7:30 on April 13 to which the members of the Honolulu County Medical Society will be invited as guests of the Hawaii Chapter of the Academy of General Practice. A cocktail-reception and buffet supper at Oahu Country club on Tuesday, April 14, will conclude the Invitational Scientific Congress.

Registration for visiting doctors will be $25.00. Registration for Hawaii doctors will be waived. However, all doctors attending the morning sessions will be expected to pay $2.50 each day to cover the cost of the breakfast. Tickets for the reception at the Oahu Country Club are $7.50 each.

Co-chairmen for the meeting are Dr. R. Varian Sloan and Dr. Homer Benson. Because a record attndance is expected, early reservations are recommended.

DRUGGISTS' CONVENTION

Dr. Troy Daniels, Dean of the School of Pharmacy at the University of California, will be the keynote speaker at the Hawaii Druggists' Convention, May 25 to 28, at the Princess Kaiulani Hotel. He will talk on the treatment of mental disease with tranquilizers, other new drugs, and psychic energizers, and the current status of oral antidiabetic preparations.

CORRESPONDENCE

(Continued from page 313)

Does the split fee encourage unnecessary referrals? For the private practitioner this is doubtful. For the group physician perhaps yes. I am sure you have heard the story of the patient with a common cold goes to a group doctor and finds himself referred to the radiologist for a chest x-ray because he has a cough, to the EENT man because his throat is sore and to the ophthalmologist because his eyes are runny. His common cold costs him $50.00 whereas in a private practitioner's office it would cost him $3.00.

Please do not construe this as a defense of fee splitting or as an attack on group medicine. We live in an era where the economic front of medical practice is expanding as rapidly and in as many different directions as is the scientific front, and I believe that group practice is as much here to stay as is closed panel practice or insurance plans, or any of the other various forms of medical practice. This is only a plea that we use with care such multi-

(Continued on page 428)

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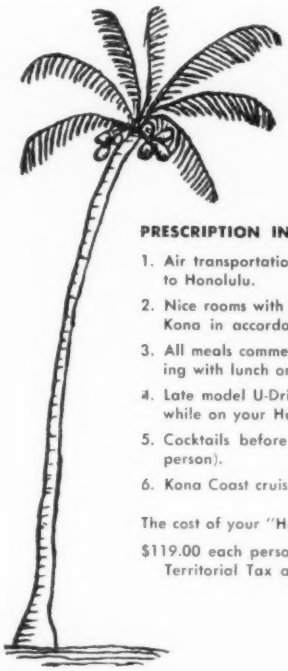
Sure-Fire
Prescription—



TAKE

HUKILAU HOLIDAY

BEFORE OR AFTER
YOUR CONVENTION

PRESCRIPTION INCLUDES:

1. Air transportation Honolulu to Hilo and Kona to Honolulu.
2. Nice rooms with private bath in both Hilo and Kona in accordance with itinerary.
3. All meals commencing lunch first day and ending with lunch on fifth day.
4. Late model U-Drive car with unlimited mileage while on your Hukilau Holiday.
5. Cocktails before dinner each evening (2 per person).
6. Kona Coast cruise on the "Captain Cook".

The cost of your "Hukilau Holiday" will be:

$119.00 each person (two in a room) plus $4.17 Territorial Tax and $3.30 Federal Airline Tax

$99.00 for third person in room plus $3.47 Territorial Tax and $3.30 Federal Airline Tax. Territorial Tax and $3.30 Federal Airline Tax.

$169.50 for single room occupancy plus $5.93 Territorial Tax and $3.30 Federal Airline Tax.

A deposit of $36.30 per person is necessary at time of making reservation—balance is payable to Kona Hukilau at conclusion of trip.

EXTRA BONUS!

When two couples or four persons travel together an exciting deep-sea fishing trip will be furnished at NO additional cost aboard one of Kona's famous fishing boats. Hours of excitement will be yours trying to hook a marlin, mahimahi, ono or some other big-game fish.

P.S. Be sure to bring along your wife.

Hukilau Resorts

HILO AND KONA

Winthrop Laboratories introduces
the first true
*"TRANQUILAXANT"**

Trancopal®

BRAND OF CHLORMETHAZANONE

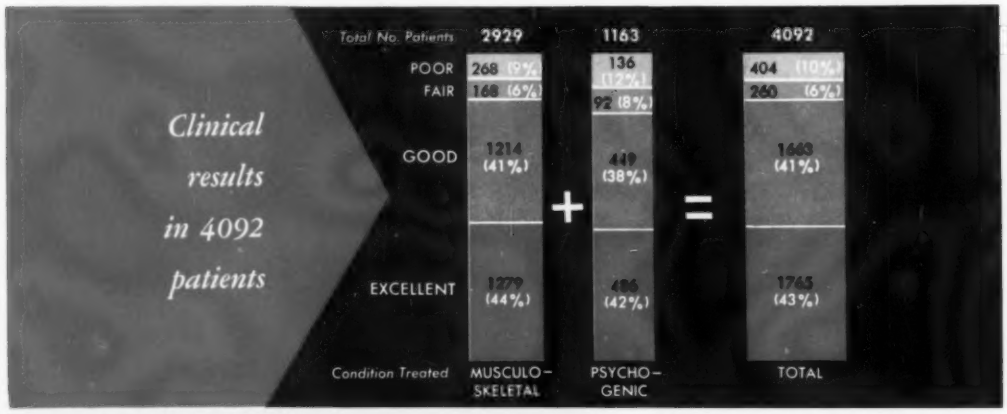
a completely new major chemical contribution to therapeutics... unrelated to any other drug in current use

- Both a muscle relaxant and a calmative agent.
- In musculoskeletal disorders, 91 per cent effective.
- In anxiety and tension states, 93 per cent effective.
- Lower incidence of side effects than with zoxazolamine, methocarbamol or meprobamate.
- No known contraindications. Blood pressure, pulse rate, respiration and digestive processes unaffected by therapeutic dosage. No effect on hematopoietic system or liver and kidney function.
- Low toxicity. In animals, even less toxic than aspirin.
- No gastric irritation. Can be taken before meals.
- No clouding of consciousness, no euphoria or depression.
- No perceptible soporific effect, even in high dosage.

designed to be equally effective as both
a MUSCLE RELAXANT
a TRANQUILIZER

*tran-qui-lax-ant (tran'kwi-lak'sant)
[< L. *tranquillus*, quiet;
L. *laxare*, to loosen, as the muscles]

Clinical results in 4092 patients



INDICATIONS:

MUSCULOSKELETAL
- LOW BACK PAIN (lumbago)
- NECK PAIN (torticollis)
- BURSITIS
- RHEUMATOID ARTHRITIS
- OSTEOARTHRITIS
- DISC SYNDROME
- FIBROSITIS
- JOINT DISORDERS (ankle sprain, tennis elbow, etc.)
- MYOSITIS
- POSTOPERATIVE MYALGIAS

PSYCHOGENIC
- ANXIETY AND TENSION STATES
- DYSMENORRHEA
- PREMENSTRUAL TENSION
- ASTHMA
- EMPHYSEMA
- ANGINA

NEUROLOGIC
- MUSCLE SPASM in paralysis agitans, multiple sclerosis, hemiplegia, poliomyelitis

DOSAGE: One Caplet (100 mg.) orally three or four times daily. Relief of symptoms occurs in fifteen to thirty minutes and lasts from four to six hours.

SUPPLIED: Trancopal Caplets® (scored) 100 mg., bottles of 100.

Winthrop Laboratories, N. Y. 18, N. Y.

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more than tetracycline alone

MYSTECLIN-V CONTAINS TETRACYCLINE PHOSPHATE COMPLEX FOR A DIRECT ATTACK ON THE PRIMARY INFECTION

Mysteclin-V strikes directly at all tetracycline sensitive organisms—most pathogenic bacteria, certain large viruses, Endamoeba histolytica. It provides all benefits of tetracycline in the effective phosphate complex form.[1] Patient response is rapid because initial high peak blood serum levels may be maintained easily at the antibacterial attack level until the infection is conquered.

MYSTECLIN-V CONTAINS MYCOSTATIN FOR A SPECIFIC DEFENSE AGAINST SECONDARY MONILIAL SUPERINFECTION

Mysteclin-V protects patients against antibiotic induced intestinal moniliasis and its complications, including vaginal and anogenital moniliasis. This protection is provided by Mycostatin, the antifungal antibiotic, with specific action against Candida (Monilia) albicans.[2]

BOTH ARE OFTEN NEEDED WHEN BACTERIAL INFECTION OCCURS

MYSTECLIN-V

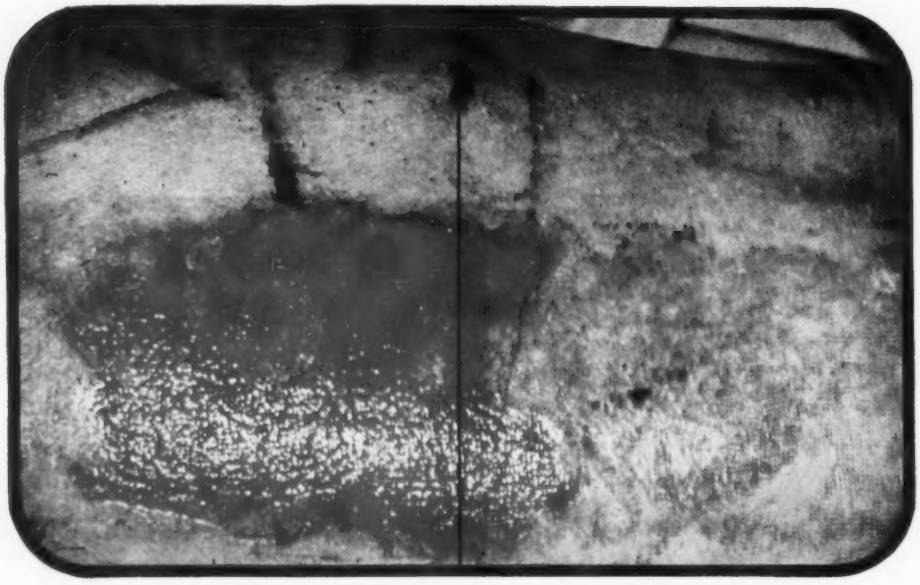
SQUIBB TETRACYCLINE PHOSPHATE COMPLEX (SUMYCIN) AND NYSTATIN (MYCOSTATIN)

Capsules (250 mg./250,000 u), bottles of 16 and 100.
Half-strength Capsules (125 mg./125,000 u), bottles of 16 and 100.
Suspension (125 mg./125,000 u per 5 cc.), 2 oz. bottles.
Pediatric Drops (100 mg./100,000 u per cc.), 10 cc. dropper bottles.

References: 1. Crunk, G. A., Naumann, D. E., and Casson, K.: Antibiotics Annual 1957-1958, New York, Medical Encyclopedia Inc. 1958, p. 397. 2. Newcomer, V. D.; Wright, E. T., and Sternberg, T. H.: Antibiotics Annual 1954-1955, New York, Medical Encyclopedia Inc., 1955, p. 686.

SQUIBB Squibb Quality—the Priceless Ingredient

*MYSTECLIN®, *SUMYCIN®, AND *MYCOSTATIN® ARE SQUIBB TRADEMARKS



Skin graft donor site after 2 weeks' treatment with ...

petrolatum gauze—still largely granulation tissue | FURACIN gauze—completely epithelialized

OBJECTIVE EVIDENCE OF EXCELLENT WOUND HEALING

was obtained in a quantitative study of 50 donor sites, each dressed half with FURACIN gauze, half with petrolatum gauze. Use of antibacterial FURACIN Soluble Dressing, with its water-soluble base, resulted in rapid and complete epithelialization. No tissue maceration occurred in FURACIN-treated areas; no sensitization was reported.

Jeffords, J. V., and Hagerty, R. F.: Ann. Surg. *145*:169, 1957.

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a broad-range bactericide that is *gentle to tissues*

spread FURACIN Soluble Dressing: FURACIN 0.2% in water-soluble ointment-like base of polyethylene glycols.

sprinkle FURACIN Soluble Powder: FURACIN 0.2% in powder base of water-soluble polyethylene glycols. Shaker-top vial.

spray FURACIN Solution: FURACIN 0.2% in liquid vehicle of polyethylene glycols 65%, wetting agent 0.3% and water.

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Nitrofurans—a NEW *class of antimicrobials—neither antibiotics nor sulfonamides*

Fiber of skeletal muscle in spasm

Fiber of skeletal muscle relaxed *(photomicrographs)*

CLINICALLY PROVEN –prolonged relaxation of acute skeletal muscle spasm

Robins

Robaxin®

Methocarbamol Robins U.S. Pat. No. 2770649 TABLETS

Summary of six published clinical studies:

ROBAXIN BENEFICIAL IN 92.4% OF SKELETAL MUSCLE SPASM CASES

	NO. PATIENTS	RESPONSE			
		"marked"	moderate	slight	none
Carpenter[1]	33	26	6	1	—
		"pronounced"			
Forsyth[2]	58	37	20	—	1
		"good"			
Lewis[3]	38	25	6	—	7
		"excellent"			
O'Doherty & Shields[4]	17	14	2	1	0
		"significant"			
Park[5]	30	27	—	2	1
		"gratifying"			
Plumb[6]	60	55	—	—	5
TOTALS	236	184 (78.0%)	34 (14.4%)	4	14

- **Highly potent—and long acting.**[1,2,3]
- **Relatively free of adverse side effects.**[1,2,3,5,6]
- **In ordinary dosage, does not reduce muscle strength or reflex activity.**[1]

REFERENCES: 1. Carpenter, E. B.: Southern M. J. 51:627, 1958. 2. Forsyth, H. F.: J.A.M.A. 167:163, 1958. 3. Lewis, W. B.: California Med. 90:26, 1959. 4. O'Doherty, D. S., and Shields, C. D.: J.A.M.A. 167:160, 1958. 5. Park, H. W.: J.A.M.A. 167:168, 1958. 6. Plumb, C. S.: Journal-Lancet 78:531, 1958.

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Ethical Pharmaceuticals of Merit since 1878

CORRESPONDENCE

(Continued from page 422)

ordinal terms (Science and Sanity, Korzybski, 3rd edition, 1948) as "closed panel" "group practice," "captive patients," "free choice of physician," etc. I sincerely hope that you will see fit to share these thoughts with the members of your group and perhaps with the medical profession at large. Our problems will not be solved by adopting the attitude of the yokel who on seeing for the first time a giraffe indignantly exclaimed, "There ain't no such animal!"

VERNON BOIDO, M.D.

Dec. 4

* * *

There is no "confusion" with reference to the "principle of aggregates." You are right in saying that if a great majority of solo practitioners indulged in fee splitting, then it would be unrealistic for the A.M.A. to oppose it on ethical grounds, or any other grounds. "Fifty million Frenchmen can't be wrong!"

The unrealism of the A.M.A.'s opposition to this practice by groups is not the basis for our objection to their opposition. We object to it on the ground that unequal division of income by physicians practicing in a group has been found to make for competition for patients within the group, and makes for reluctance to ask for consultation.

Finally, you bring up a point which indicates a misunderstanding about group practice. A basic rule in group practice is "one fee for one visit for one illness." If the internist wants his patient with a cold examined by the EENT man, the latter makes no charge. If he wants his chest x-rayed, there is a charge; but surely your hypothetical $3 office visit to the GP would not include a chest film? Even if five or six different specialists must see the patient, only one of them makes the office visit charge. The problem is not really to avoid overcharging the patient—the problem is to avoid overworking the partnership in the process of earning the fee. And referral outside the group is done with alacrity if the patient so much as suggests it.

—EDITOR

HONOLULU COUNTY MEDICAL LIBRARY

(Continued from page 382)

with the architect and at various times with The Queen's Hospital Board and that all concerned were kept well informed. The contract with the architect had been drawn and was agreeable and favorable to both parties. Mr. Ossipoff has been given a list of our requirements, and the preliminary plans are expected momentarily. Dr. Lowrey asked about the display of the plans, and there was some discussion of this matter. It was agreed that they should be given the widest circulation possible. The Building Committee hopes that comments will be forthcoming from the membership.

NOMINATING COMMITTEE REPORT Dr. Kobayashi reported that the Nominating Committee had renominated the following incumbents to serve for a period of three years: Drs. Ando, Batten, Gebauer, and Lum. He explained that maintaining the continuity of the Board seemed important at this time and that the meeting was open to nominations from the floor. He also read the names of other members of the Board whose terms expire

(Continued on page 432)

REMEMBER!

The next time you get a prescription from your eye physician (M.D.), take it where you can be assured of first quality lenses. A large and beautiful selection of frames, accurate fitting and superior servicing.

SEE YOUR
GUILD OPTICIAN

GUILD OPTICIANS
OPTICAL DISPENSERS
of Hawaii
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Have a Coke!

DRINK Coca-Cola

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SIGN OF GOOD TASTE

Bottled Under Authority of The Coca-Cola Co. by
The Coca-Cola Bottling Co. of Honolulu, Ltd.

If one . . . or all . . . needs nutritional support . . .

they
deserve

GEVRAL®

capsules—14 VITAMINS AND 11 MINERALS

Vitamin-Mineral Supplement Lederle

For Complete Formula see PDR (Physicians' Desk Reference), page 689

LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

Lederle

In potentially-serious infections...

[illegible]

The Upjohn Company, Kalamazoo, Michigan

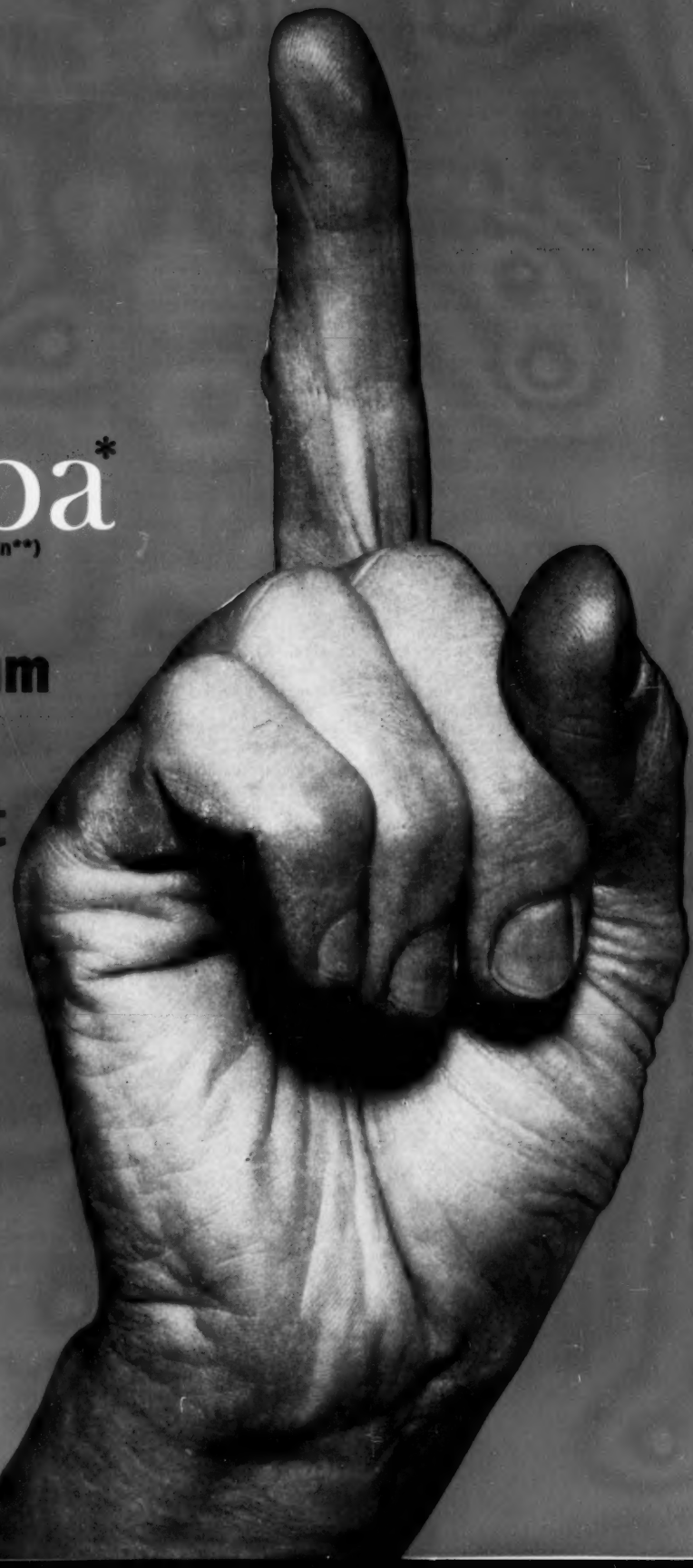
Upjohn

Make new Panalba*

(Panmycin† Phosphate plus Albamycin**)

your broad-spectrum antibiotic of first resort

effective against more than 30 common pathogens, even including resistant staphylococci.



Available forms:

1. Panalba Capsules, bottles of 16 and 100 capsules. Each capsule contains:

Panmycin phosphate (tetracycline phosphate complex) equivalent to tetracycline hydrochloride ..... 250 mg.
Albamycin (as novobiocin sodium) ... 125 mg.

2. Panalba KM,†† Flavored Granules, 60 cc. size bottle. When sufficient water is added to fill the bottle, each teaspoonful (5 cc.) contains:

Panmycin (tetracycline) equivalent to tetracycline hydrochloride ..... 125 mg.
Albamycin (as novobiocin calcium) .. 62.5 mg.
Potassium metaphosphate ..... 160 mg.

Dosage:

Panalba Capsules. Usual adult dosage is 1 or 2 capsules 3 or 4 times a day.

Panalba KM Granules

For the treatment of moderately acute infections in infants and children, the recommended dosage is 1 teaspoonful per 15 to 20 lbs. of body weight per day, administered in 2 to 4 equal doses. Severe or prolonged infections require higher doses. Dosage for adults is 2 to 4 teaspoonfuls 3 or 4 times daily, depending on the type and severity of the infection.

HONOLULU COUNTY MEDICAL LIBRARY

(Continued from page 428)

December 31, 1959: Drs. Gotshalk, Kobayashi, Moffat, and Dean Walker; and those with expiring terms of December 31, 1960: Drs. Civin, Lowrey, Min, and Morgan.

Dr. West moved that nominations be closed and the motion was seconded. He also moved that the Secretary cast a unanimous ballot in favor of the four nominees and that they be duly elected. The motion was seconded and passed.

Dr. Lowrey remarked that it was disappointing that so few members of the Library were present at their Annual Meeting. He suggested sending out copies of the reports so that everyone be informed. Various methods of circulating the reports were discussed, and it was agreed that they be published in the HAWAII MEDICAL JOURNAL and also mimeographed for mailing if costs were not prohibitive.

After some further discussion of fund-raising problems, the meeting adjourned.

(MRS.) ETHEL HILL, *Librarian*

COUNTY SOCIETY REPORTS

(Continued from page 388)

equities. Dr. Mitchel pointed out that Dr. Cheim has requested a meeting where he could talk to the doctors about V.A. It was pointed out that this matter applies only to the doctors of the Hilo Memorial Staff, and it was suggested that it should be undertaken as a Hilo Memorial Staff problem.

The business portion of the meeting was then brought to a close at 8:30 P.M. Dr. Okada again introduced Dr. White who gave a slide-introduced talk on the subject of trauma. Dr. White then introduced Colonel Ernest Lineberger, Chief of the Orthopedic Department of Tripler Army Hospital who gave a slide-illustrated talk covering the over-all management of trauma cases at Tripler Army Hospital. The meeting was brought to a close at 10:00 P.M.

ED B. HELMS, M.D.
Secretary

Maui

The regular meeting of the Maui County Medical Society was called to order by President Dr. Joseph E. Andrews on Tuesday, November 18, 1958, at the Central Maui Memorial Hospital.

Dr. David Kliewer gave a very interesting talk on "Hemolytic Diseases of Newborns." Many questions were asked after his presentation.

L. T. KASHIWA, M.D.
Secretary

Honolulu

The Honolulu County Medical Society met on Tuesday, September 2, 1958, at 7:45 P.M. Dr. R. T. West presided and approximately 85 members were present.

Dr. Hartwell, Chairman of the Indoctrination Committee, introduced the following new members: Drs. Stanley E. Kobashigawa, Paul L. Leyda, Joseph T. Smith, Calvin Sia, and Grant Stemmermann.

Dr. John Lowrey presented a report on the Annual Blue Shield Conference in Chicago. Dr. Buzaid moved that Dr. Lowrey's remarks be circulated to the membership and also printed in the HAWAII MEDICAL JOURNAL. The motion was seconded and was passed.

Dr. O. D. Pinkerton and Dr. C. C. McCorriston presented a complete report on the Medicare Program to date and brought the Society up to date on the recent negotiations with the government for a new contract. Dr. Pinkerton showed six slides to illustrate local utilization of the program and the new proposed rules on the curtailment of the program. Questions from the floor were answered by Drs. Pinkerton and McCorriston. Admiral Clay, Fleet Surgeon, presented his views on the program, and stated that they were very interested in seeing that the program worked out well for their dependents.

A report on the Medical Library's Fund Raising Drive was presented by Dr. Lowrey. He stated that to date $19,000.00 has been received from our local doctors. The committee is now approaching local foundations. etc. Dr. Lowrey urged all doctors to talk this program up.

Dr. Arnold, Jr., presented a report on the AMA convention which he attended as a delegate. He urged all members to make their contributions towards Medical Education through the AMEF rather than contribute directly to the school. He stated that you can donate through the AMEF and have it sent to the school of your choice.

Dr. Quisenberry spoke on the AMA's stand on United Fund Drives and stated that he felt this Society should go on record as supporting the action of the House of Delegates of the AMA on United Fund Drives.

(Continued on page 434)

"PERSONAL FLUORIDATION" "PERSONAL FLUORIDATION" "PERSONAL FLUORIDATION" "PERSONAL FLUORIDATION"

"PERSONAL FLUORIDATION"

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establishes peaceful indifference to preoperative preparation *without serious hypotensive effects.*

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makes possible the maintenance of an adequate degree of narcosis with reduced doses of narcotics.

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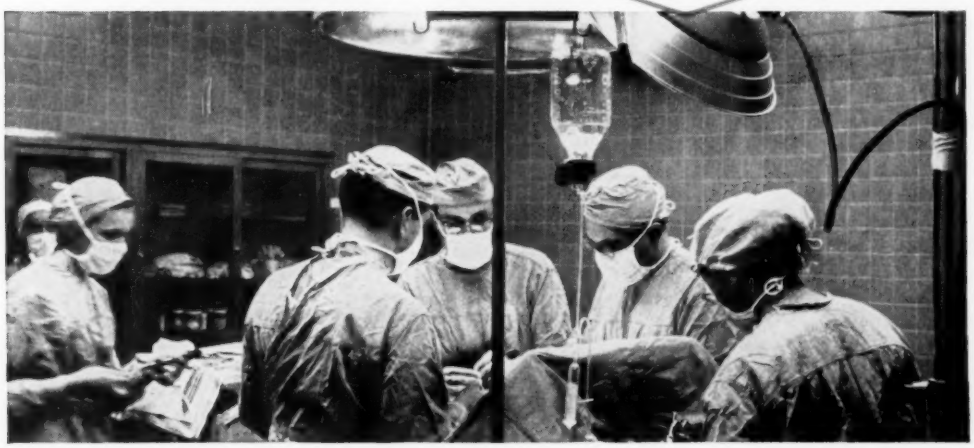

COUNTY SOCIETY REPORTS

(Continued from page 432)

The report of the Fee Adjustment Committee concerning changes in the Relative Value Schedule, which had been circulated to the membership, was accepted with the following changes: Procedure 3315 unit value changed to 15.0, and Procedure 3317 unit value changed to 20.0.

A first reading of amendments to the Constitution and Bylaws was presented by Dr. West who explained briefly each amendment as he went along. These amendments will be circulated to the membership ten days before the next meeting at which time it will be brought up for discussion and approval.

Dr. Marnie introduced a resolution to permit Dr. Amlin to reapply for membership in the County Medical Society before the expiration of one year. In accordance with our Bylaws a copy of this resolution will be circulated to the membership 10 days before the next meeting at which time it will be acted upon.

There being no further business, the meeting was adjourned to the lanai for refreshments.

* * *

The Honolulu County Medical Society met on Tuesday, October 7, 1958, at 7:30 P.M. Dr. R. T. West presided and approximately 98 members were present.

Mr. R. M. Kennedy, Executive Secretary of the Society, presented a talk on "Medical Economics." Much of his talk dealt with the Bureau of Medical Economics. He reviewed the Bureau's operations which he illustrated with slides to give a clearer picture of what happens when an assignment is received from a doctor and how the account is worked. He then discussed in detail several additional services to be offered by the Bureau which included a "physician's retirement fund," an "employment agency," and a "billing department" to handle the accounts receivable of doctors—a means of cutting down on a doctor's office overhead. In closing Mr. Kennedy invited the doctors to visit the Bureau following the meeting and stated that Mr. Short, the Bureau's manager, would be there to answer any questions.

Dr. Pershing Lo of the Indoctrination Committee introduced the following new members: Drs. Merven Chun, Charles C. Custer, Thomas K. L. Lau, and Philip J. W. Lee.

Resolutions in memory of Dr. Virgil A. Harl and Dr. Rolla O. Brown were read by Dr. Beck, Chairman of the Resolutions Committee. A minute of standing silence in their memory was observed by the membership.

Dr. Batten reported on the Library's Building Fund, which to date amounts to $68,000.00. He urged those doctors who have already contributed, to contribute a little more if possible, and those doctors who hadn't at all to make some contribution, either as an outright donation or in the form of a pledge.

Dr. West reread the resolution that was presented at the last membership meeting which proposed that Dr. Kenneth Amlin be given the privilege of reapplying for membership in the Honolulu County Medical Society before the expiration of one year. Dr. West stated that in accordance with our Bylaws, the resolution was circulated to the membership ten days before this meeting and that it will be voted on by secret ballot. Following a count of the ballots, Dr. West announced that the resolution had failed to carry, 57 to 36. Two were blanks. 95 ballots were cast.

(Continued on page 438)

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more hypertensives can be better controlled
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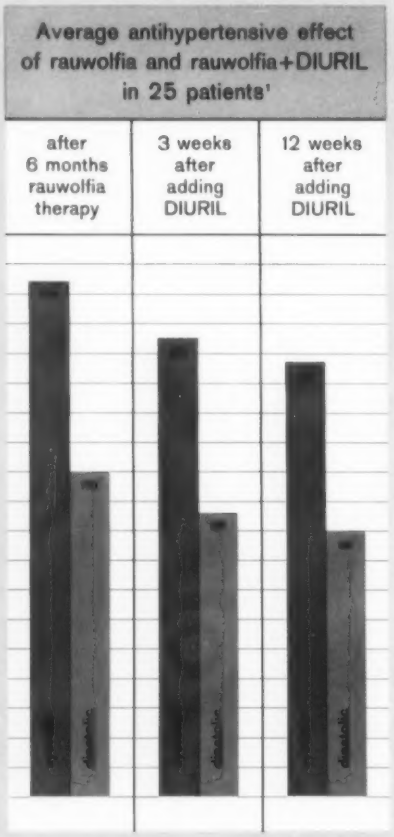
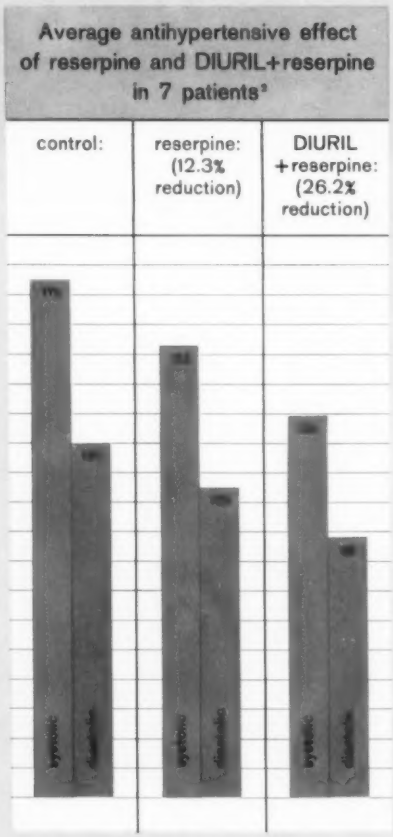
potentiated effect

DIUPRES produces an effect greater than either DIURIL or reserpine alone. It is effective in many patients who respond inadequately or not at all to either DIURIL or reserpine.


Average antihypertensive effect of rauwolfia and rauwolfia+DIURIL in 25 patients[1]
after 6 months rauwolfia therapy
3 weeks after adding DIURIL
12 weeks after adding DIURIL


Average antihypertensive effect of reserpine and DIURIL+reserpine in 7 patients[2]
control:
reserpine: (12.3% reduction)
DIURIL +reserpine: (26.2% reduction)

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effective therapy for most patients

DIUPRES by itself usually provides effective therapy for a majority of patients with mild or moderate hypertension, and even for many patients with severe hypertension. Many patients now treated with other agents which frequently cause distressing side effects can be adequately managed with well tolerated DIUPRES.

provides basic therapy

Should other drugs need to be added to DIUPRES, they can be given in much lower than usual dosage so that their side effects are often strikingly reduced.

rapid onset of effect

The antihypertensive action of DIUPRES is rapidly evident. (Considerable time may elapse before the antihypertensive effect of reserpine alone is observed.)

fewer and less severe side effects

DIUPRES may be expected to cause fewer and less severe side effects than are encountered with other antihypertensive therapy. (Since DIURIL and reserpine potentiate each other, the required dosage of each is usually less when given together as DIUPRES than when given alone. Such reduction in dosage makes side effects less likely to occur.)

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DIUPRES minimizes the problem of weight gain seen with reserpine (reserpine alone has been reported to produce weight gain in 50 per cent of patients).[1,4]

virtually eliminates fluid retention

DIUPRES is not likely to cause either clinical or subclinical retention of sodium and water. (Hypotensive drugs, particularly rauwolfia[5] and hydralazine,[6] may cause fluid retention. Even when such retention is subclinical, their antihypertensive effectiveness is diminished.[6])

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> *"It may well be that the drug* [DIURIL] *produces the benefits of a markedly restricted low sodium diet but without its hardships."*[3]

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DIUPRES allays anxiety and tension, thus reducing the emotional component of hypertension. Organic changes of hypertension may be arrested and reversed. Headache, dizziness, palpitations and tachycardia are usually promptly relieved by DIUPRES. When the *anginal syndrome* accompanies hypertension, the administration of DIUPRES may also cause diminution or even disappearance of this syndrome concurrent with control of the hypertension.

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Indications:
DIUPRES is indicated in hypertension of all degrees of severity. It can be used in the following ways:

- as total therapy
- as primary therapy, adding other drugs if necessary
- as replacement or adjunctive therapy in patients now treated with other agents

Precautions:
The precautions normally observed with DIURIL or reserpine apply to DIUPRES. Additional information on DIUPRES is available to physicians on request.

Recommended dosage range:
DIUPRES-500—one tablet one to three times a day.
DIUPRES-250—one tablet one to four times a day.
If necessary, other agents may be added.
If the patient is receiving ganglion blocking agents or hydralazine, their dosage should be cut by 50 per cent when DIUPRES is added.

Diupres-500

DIUPRES-500

500 mg. DIURIL (chlorothiazide), 0.125 mg. reserpine.
Bottles of 100, 1000.

Diupres-250

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1. Rochelle, J. B., III, Bullock, A. C., and Ford, R. V.: Potentiation of antihypertensive therapy by use of chlorothiazide, *J.A.M.A.* 168:410, Sept. 27, 1958. 2. Freis, E. D., Wanko, A., Wilson, I. M., and Parrish, A. E.: Treatment of essential hypertension with chlorothiazide (Diuril), *J.A.M.A.* 166:137, Jan. 11, 1958. 3. Freis, E. D.: Treatment of hypertension. (Presented at the Annual Meeting of Southern Medical Association, Nov. 13, 1957.) 4. Moyer, J. H., Dennis, E., and Ford, R.: Drug therapy (Rauwolfia) of hypertension, *A.M.A. Arch. Int. Med.* 96:530, Oct. 1955. 5. Perera, G. A.: Edema and congestive failure related to administration of rauwolfia serpentina, *J.A.M.A.* 159:439, Oct. 1, 1955. 6. Wilkins, R. W.: Precautions in use of antihypertensive drugs, including chlorothiazide, *J.A.M.A.* 167:801, June 14, 1958.

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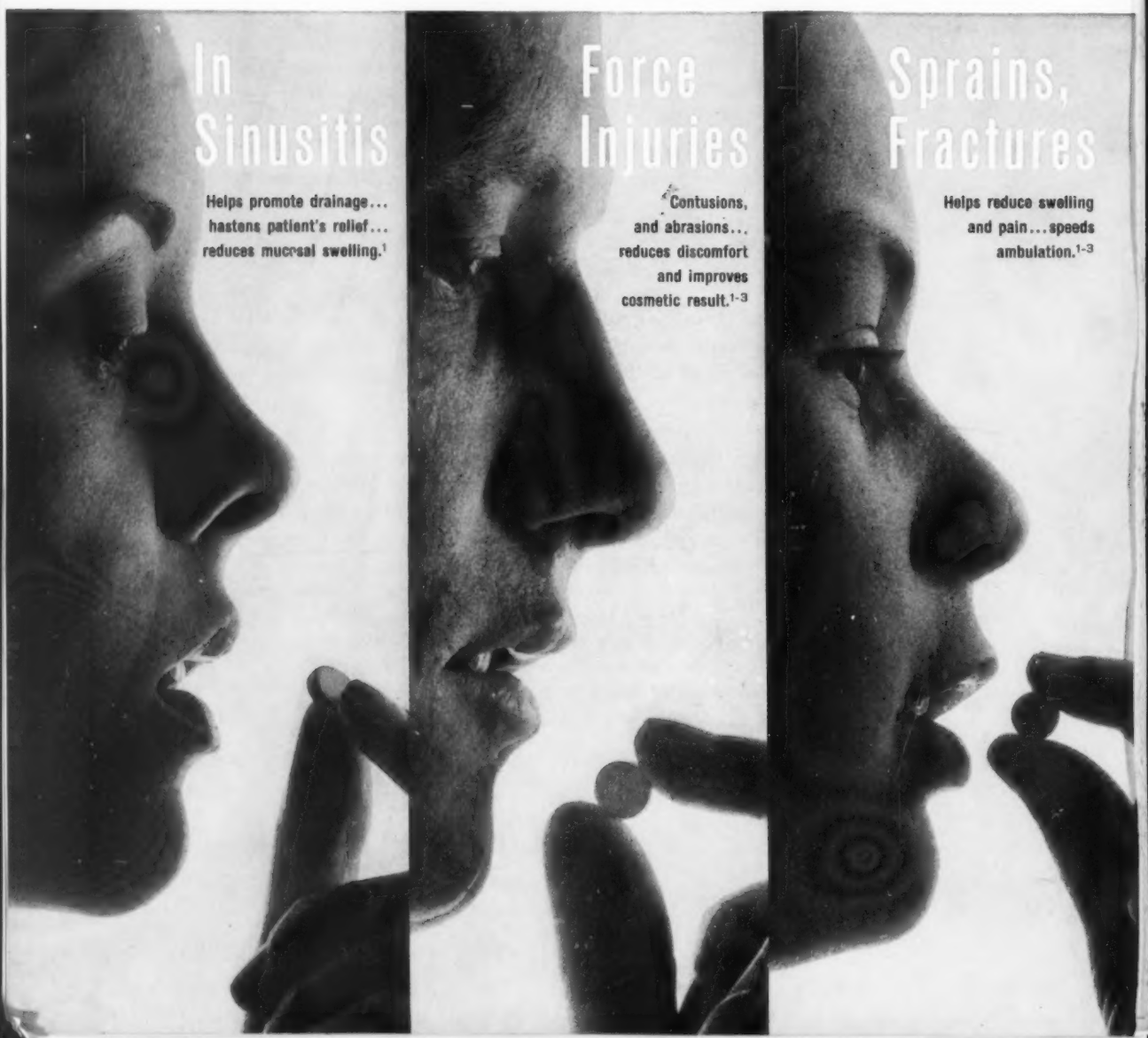
References: 1. Innerfield, I.; Shub, H., and Boyd, L. J.: New England J. Med. 258: 1069 (May 24) 1958. 2. Miller, J. M.; Godfrey, G. C.; Ginsberg, M. J., and Papastrat, C. J.: J. A. M. A. 166:478 (Feb. 1) 1958. 3. Davidson, E; Prigot, A., and Maynard, A. de L.: Harlem Hosp. Bull. II: 1 (June) 1958 *Reg. U. S. Pat. Off.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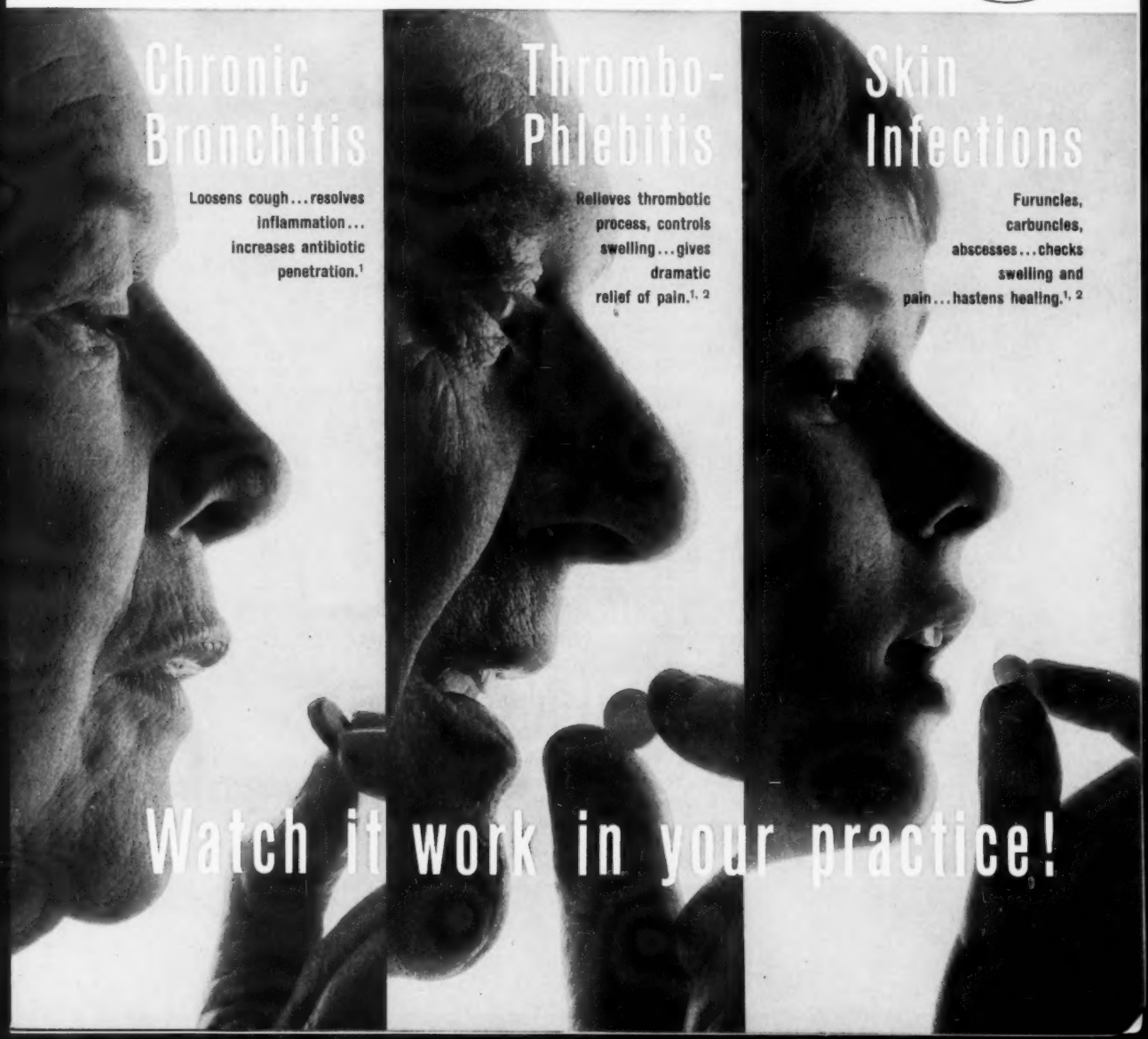
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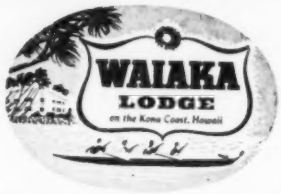

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COUNTY SOCIETY REPORTS

(Continued from page 434)

Dr. West announced the following actions taken by the Board of Governors at their last meeting:

1. Appointment of Dr. Vasconcellos and Dr. Yokoyama to the Committee of the Prevention of Rheumatic Fever of the Hawaii Heart Association.
2. Approved the earmarking of $30,000.00 from Operation Hypo for the Honolulu County Medical Library's building fund.
3. The Honolulu County Medical Society and the Hawaiian Academy of Science will co-sponsor a talk by Dr. Andrew Ivy, Sr., on "Atherosclerosis in Humans."
4. The Honolulu County Medical Society and the Hawaii Heart Association will co-sponsor a reception for three past presidents of the American Heart Association at the Philip Spalding's home on November 1.
5. Approved a total of $215.00 to be used as prizes for local students in the A.A.P.S. Essay Contest sponsored by the Woman's Auxiliary of the County Medical Society.

Amendments to the Constitution and Bylaws which had been circulated to the membership 10 days prior to this meeting were brought for approval. Dr. West stated that each amendment will be taken up individually and discussed, and if there are no objections to the amendment, he would consider the amendment approved. He mentioned that these amendments had been approved by the Board of Governors and that it would require a ⅔'s vote of the membership present to pass the amendments. Amendments 1 to 44 were then taken up and were approved with the exception of ten amendments which were discussed and voted on.

There being no further business the meeting was adjourned to the lanai for refreshments at 10:45 P.M.

H. Q. PANG, M.D.
Secretary

BOOK REVIEWS

(Continued from page 383)

quality. The currently popular tumor atlases are not superior to this volume in this regard. It is probable that for years to come this will be the most important reference work in this field.

Space restrictions do not permit the extensive review that each chapter of this work demands. Your reviewer, however, was especially taken by the succinct condensation of lesions peculiar to jaw bones and the chapter on radiation injury of bone. The author's observation that irradiation of otherwise benign bone tumors may stimulate malignant change serves to emphasize the importance of an adequate pathologic appraisal of all tumorous lesions of bone.

GRANT N. STEMMERMANN, M.D.

Orthopedics for the General Practitioner

By William E. Kenney, M.D., and Carroll B. Larson, M.D., F.A.C.S., 413 pp., illus., $11.50, The C. V. Mosby Company, 1957.

The book might better have been titled *Orthopedics for the Medical Student.* It is an outline of orthopedic and traumatic surgery condensed into 400 pages, and will be of little value to the general practitioner. An adequate, up-to-date bibliography after each chapter would have increased the value. Instead, there is only a list of 21 references, some of them standard textbooks, some of them outdated and of historical value only.

Chapter one on Diseases or Affections in Childhood

(Continued on page 444)

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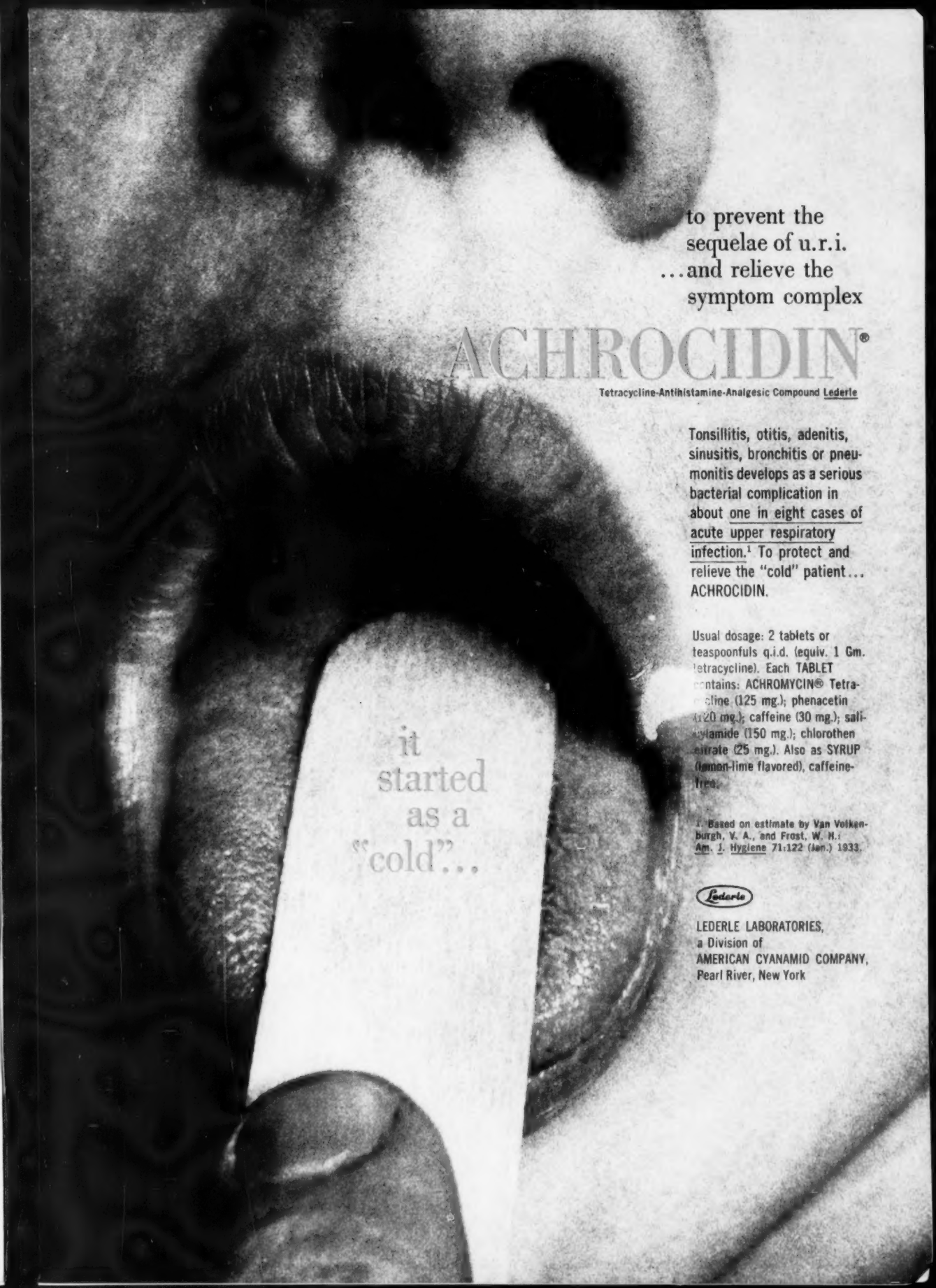

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1. Based on estimate by Van Volkenburgh, V. A., and Frost, W. H.: Am. J. Hygiene 71:122 (Jan.) 1933.

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1. Landes, R. P. and Peters, M.
Postgrad. Med. 23:648, June 1958.

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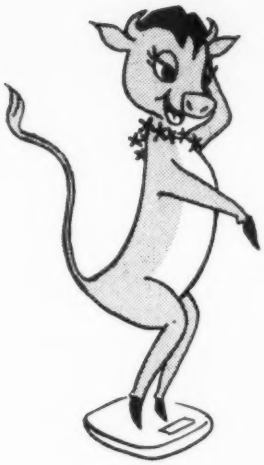

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(Continued from page 438)

is the best part of the book and warrants reading, particularly by pediatricians.

WILLIAM H. GULLEDGE, M.D.

Also Received

Water and Electrolyte Metabolism in Relation to Age and Sex—Vol. 4

By G. E. W. Wolstenholme, O.B.E., M.A. M.B., B.Ch., and Cecilia M. O'Connor, B.Sc., 327 pp., $8.50, Little, Brown and Company, 1958.

Informative and authoritative, but not for light reading.

★ Speech Recovery After Total Laryngectomy

By C. J. Hodson, F.R.C.P., D.M.R.E., F.F.R., and Miss M. V. O. Oswald, F.C.S.T., 36 pp., $1.50, The Williams & Wilkins Company, 1958.

Otolaryngologists need this little pamphlet for instructing their laryngectomees.

Cold Injury, Ground Type

By Colonel Tom F. Whayne, M.C., U.S.A. (Ret.), and Michael E. DeBakey, M.D., 570 pp., $6.25, Office of the Surgeon General, Department of the Army, Washington, D. C., 1958.

An exhaustive treatise on frostbite.

Cerebral Vascular Diseases

By The American Heart Association, Princeton, New Jersey; January 16-18, 1957, Irving S. Wright, Chairman, Clark H. Millikan, Editor, 224 pp., $4.00, Grune & Stratton, Inc., 1958.

Forty medical leaders from all over the world discuss all aspects of "strokes," as of January, 1957. Practical and authoritative.

★ The Physical Treatment of Varicose Ulcers

By R. Rowden Foote, F.I.C.S., M.R.C.S., L.R.C.P., D.R.C.O.G., 125 pp., $4.00, Williams & Wilkins Company, 1958.

Practical and detailed instructions for the management of varicose ulcers, with numerous good illustrations.

Trifluoperazine—Clinical and Pharmacological Aspects

By Henry Brill, M.D., 219 pp., $3.50, Lea & Febiger, 1958.

Twenty-five articles by 44 contributors, plus a bibliography of 30 more, all about one not-yet-very-widely-known (in Hawaii, anyhow) tranquilizer.

★ Heredity of the Blood Groups

By Alexander S. Wiener, A.B., M.D., F.A.C.P., F.C.A.P., and Irving B. Wexler, A.B., M.D., F.A.C.P., 150 pp., $6.00, Grune & Stratton, 1958.

Authoritative and indispensable reference work for hematologists, serologists, and those concerned with inheritance of blood group patterns.

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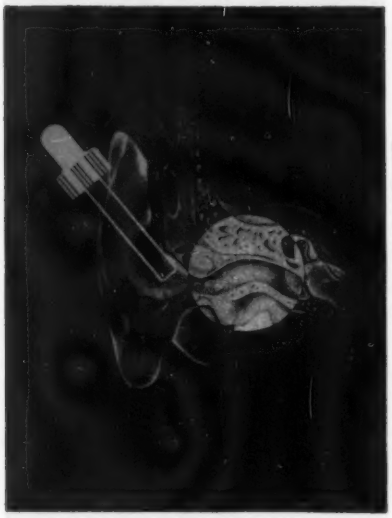

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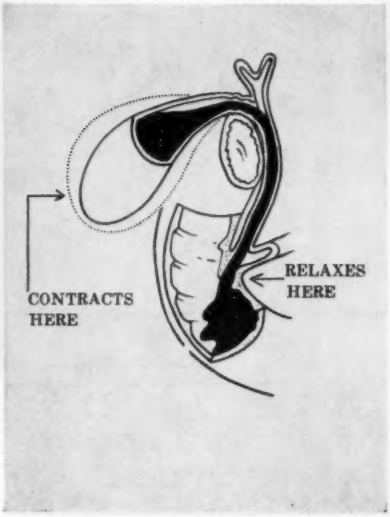

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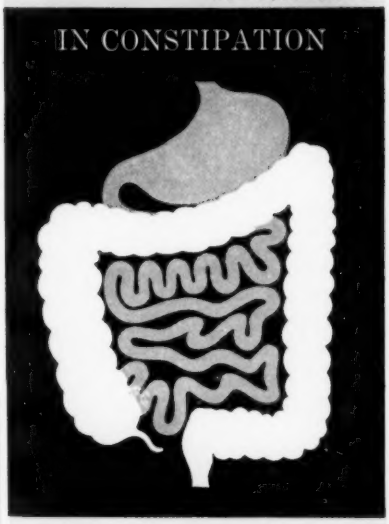

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1. Herland, A. L., Lowenstein, A.: Quart. Rev. Surg. Obst. & Gynec. 14:196 (Dec.) 1957

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	Page
Abbott Laboratories	Insert (between 408 and 409), 409, 410, 411, 412, 413
American Factors, Ltd.	444
Ames Company	328, 447
Ayerst Laboratories	332, 390, 391
Baxter, Don, Inc.	419
Boyle & Company	406, 407
Burroughs Wellcome	334, 417
Carnation Company	319
Coca-Cola Bottling Company	428
Dairymen's Association, Ltd.	443
Davies, Theo. H., & Co., Ltd.	313
Eaton Laboratories	316, 426
Ethicon, Inc.	Insert (between 328 and 329)
Geigy Pharmaceuticals	415
General Electric	323
Hawaii Camera Sales	438
Hawaii Medical Service Association	380
Hawaiian Airlines	418
Hawaiian Electric Co.	324
Home Insurance Co.	401
Hukilau Resorts	423
Inter-Island Resorts, Ltd.	438
Jeffrey, Ed	446
Lederle Laboratories	322, 336, 378, 379, 402, 403, 420, 421, 429, 436, 437, 439
Lilly, Eli & Co.	309, 338
Lorillard, P., Co.	325
Maltbie Laboratories	327
Mead Johnson International	317
Merck, Sharp & Dohme	Insert (between 434 and 435), 315, 333, 440, 441
Optical Dispensers	428
Parke, Davis & Co.	310, 311, 329
Pet Milk Co.	335
Pfizer Laboratories	330, 331, 433, 442
Purdue Frederick Co.	445
Riker Laboratories	326
Robins, A. H., Co., Ltd.	320, 321, 427
Schering Corp.	Insert (between 318 and 319), 337
Schieffelin & Co.	418
Schuman Carriage Co.	408
Searle, G. D., & Co.	389
Sears Roebuck & Co.	401
Smith, Kline & French	448
Squibb & Sons	318, 425
Star-Bulletin Printing Co., Inc.	422
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(1) Rubin, A., and Babbott, D.: J.A.M.A. *168*:498, (Oct. 4) 1958. (2) Kinsey, A. C.; Pomeroy, W. B., and Martin, C. E.: Sexual Behavior in the Human Male, Philadelphia, W. B. Saunders Company, 1948.

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